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SILICOSIS AND A FEW OF THE OTHER PNEUMOCONIOSES: OBSERVATIONS ON CERTAIN ASPECTS OF THE PROBLEM, WITH EMPHASIS ON THE ROLE OF THE RADIOLOGIST*

CALDWELL LECTURE, 1957

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Eugene Wilson Caldwell

This is the occasion of the Caldwell Lecture and before I begin my address, I want to express to our president, Dr. Wendell G. Scott and to the members of the Program Committee my sincere appreciation for the honor that they have conferred on me in asking me to present this lecture before the American Roentgen Ray Society. I did not have the privilege of knowing Dr. Caldwell, but those who did have testified on many occasions of his ideals and achievements. This Society has been most fortunate in having many who have contributed much toward the advancement of medicine in general and of radiology in particular. Our heritage is one of which we justly can be proud. I have the feeling that Dr. James T. Case and others who were responsible for the inauguration of the Caldwell Lecture, not only wished to honor Caldwell, but in addition hoped that this special event might become a symbol by which we could honor all of our members and those in collateral disciplines of investigation and in industry who during their lifetime added their bit to our specialty.

As evidence of this premise, the senior Dr. William A. Evans[18] wrote the following: "The early second period of pioneering in American roentgenology was graced by several who by virtue of their noble character and unusual professional attainments have been canonized by those who continued in the work for which these made the supreme sacrifice. One who will change the whole direction of his life current to follow an ideal conceived after comparative success in a given endeavor, must possess unusual attributes. Such was Eugene W. Caldwell of New York. Already a master mind in the field of electrical engineering and physics, upon becoming interested in Röntgen's work, he early appreciated its importance both medical and otherwise. The better to serve its advancement in the field of human relief, Caldwell qualified himself as a physician. Thus trained in two highly technical and scientific spheres, he began a career which added greatly to the efficiency and completeness of roentgen equipment and accessories and its proper adaptation for demonstrating various medical and surgical lesions."

Dr. Evans goes on and describes the work of others and concludes with the following statement: "With Leonard, these three, Caldwell, Dodd, and Kassa-

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bian, succumbed to roentgen injuries incident to their constant studies. The American Roentgen Ray Society, conscious of their beneficent influence, honored Leonard by the establishment of the 'Leonard Prize,' an award granted at intervals for meritorious studies in some phase of roentgenology and commemorates the influence of Caldwell by the 'Caldwell Lecture'. . . . "

THE subject of my address is concerned with certain aspects of silicosis and some of the other pneumoconioses. Many of you know of my interest in this problem. This is the fourth time that I have used this subject in a named lectureship—the Preston M. Hickey Memorial Lecture[65] before the Wayne County Medical Society, April 6, 1942; the Russell D. Carman Memorial Lecture[66,67] before the Minnesota State Medical Association, June 12, 1950; and the Ross Golden Lecture[69] in New York, March 21, 1955, before the New York Roentgen Society.

My own experience on the effects of dust inhalation on the lungs dates back to 1919, when following World War I, Dr. Henry K. Pancoast re-embarked on the studies which he, Dr. T. Grier Miller and Dr. H. R. M. Landis began in 1916.[62] Together, we carried out many studies over the succeeding years. Dr. Pancoast was the first radiologist in this country and one of the few in the world who early made a real effort to learn something about pneumoconiosis. In so doing, he developed many friends outside of radiology in the fields of public health, pathology, chemistry, physics, engineering, law, and industry. For me, meeting and getting to know others interested in dust hazards has been a most rewarding experience.

Anyone who takes the time to read a little about the pneumoconioses cannot help but be impressed with certain facets of the total problem. Some of these are of real importance to the radiologist. Most of the diagnoses begin with an interpretation of the roentgenograms. A radiologist with a broad background of diagnostic training and experience can be a tremendous asset in the care that these dust-affected workers receive because there are many diseases that produce shadows in the lungs that simulate silicosis and asbestosis. There is no part of radiology in which there is a greater opportunity for contributing real service to the health of the community and of the nation. The retrospective studies of chest roentgenograms made on industrial workers provide a most satisfying experience, not only in studying the natural history of the pneumoconioses, but other pulmonary and cardiac diseases.

Over the years I have talked with young men who have trained with us in an effort to stimulate interest in the field of the pneumoconioses, and only a few have responded. For the most part, radiologists have been satisfied with a casual acquaintance with the subject. The information that these men are qualified to render is oftentimes inadequate. Because of such indifference, internists, chest physicians, chest surgeons, pathologists and others are now rendering interpretation of roentgenograms in many places. We, as radiologists, have a very definite obligation in improving this situation.

GENERAL ROENTGENOLOGIC CONSIDERATIONS

The proper roentgen interpretation of pneumoconiosis is dependent on; (1) a knowledge of the anatomy of the chest and the physiologic problems associated with its anatomic constituents; (2) a thorough familiarity with normal fluoroscopic and roentgenographic appearances and their permissible variations; (3) an understanding of the histology of the lungs and especially the lymphatic system; and (4) a clear perception of the pathology of pneumoconiosis and of all conditions which may simulate its roentgenographic appearances.

In many instances, the radiologist is expected to render an opinion from a single roentgenogram with or without an adequate history. Under such circumstances, one has to do the best he can. A report

should include the conditions that should be considered in arriving at the final or presumptive diagnosis.

Although I have had the opportunity of studying roentgenograms of collective surveys done by the Public Health Service, or some other like agency, my experience has been concerned largely with three groups: (I) *The individual who arrives in a general hospital* usually with a complaint relating to some system or organ in the body other than the lung; (II) *the individual who is seeking compensation* for pneumoconiosis; and, (III) *workers in an industry where there is a known or unknown dust hazard* in certain of its operations.

I and II. The roentgen studies for the *general hospital patient* and the *individual who is seeking compensation* can be considered together, because the studies are essentially the same. These include a fluoroscopic and roentgenographic study in addition to all of the aforementioned history review, physical and laboratory examinations. It has been my practice to take a supplementary history myself, making a real effort to learn everything about the individual's total environment.

The fluoroscopic and roentgenographic studies should be planned to obtain good records of any lesions, and to demonstrate, if possible, any roentgen evidence of disability. In pneumoconiosis, one of the problems of disability has to do with pulmonary ventilation.

Ventilation is a cyclic process of inspiration and expiration in which alternately fresh air enters the respiratory tract and an equal amount of pulmonary gas is exhaled.[4] An adequate ventilation should be accomplished without an undue expenditure of energy. It is important to define several terms[13] in order to avoid confusion:

1. Tidal volume (depth of breathing) is the volume of gas inspired or expired during each respiratory cycle.
2. Residual volume is the volume of gas remaining in the lung at the end of a maximal expiration.
3. Total lung capacity is the amount of gas contained in the lung at the end of a maximal inspiration.
4. Vital capacity is the maximal amount of gas that can be expelled from the lungs by forceful effort following a maximal inspiration.

There are many anatomic abnormalities which reduce the vital capacity and which can be demonstrated in roentgenograms of the chest.[2] These pertain to: anomalies in development of vertebrae or ribs,[71] scoliosis, deformities of the thoracic cage due to fractures, thickened pleura, and space-taking lesions within the chest.[4] Lesions affecting the position and mobility of the diaphragm may reduce seriously the vital capacity.[4] Changes in the interstitial tissue caused by edema, fibrosis, or granulomas are associated sometimes with reduction in total lung volume. Roentgenograms should be made in inspiration and in expiration without changing the position of the tube. Roentgenograms in each lateral view should also be obtained.

The patient's chest should be studied fluoroscopically in the erect, horizontal, prone and supine positions with horizontal and vertical beams, and in each decubitus position during quiet and forced respiration, and after coughing and sniffing. Fluoroscopy during the Müller (sniffing against a closed glottis) and the Valsalva maneuvers (straining against a closed glottis) is informative. This gives one the opportunity to determine the movements of the thoracic cage; the position of the mediastinal structures in all projections and during all phases of respiration; the pulsation of the heart and great vessels; the presence or absence of disturbed ventilation. It is well known that obstructive emphysema sometimes is seen best during fluoroscopy.[29]

While the fluoroscopic examination during forced respiration cannot provide an absolute figure of the dynamic reserve of the lungs, one can get a good estimate of the movement of air by observing the movement of the diaphragm and thoracic cage.[4] If fluoroscopy is combined with vital

capacity spirometry, the radiologist will improve his clinical judgment.[4] If the patient performs a maximal breathing capacity test, and there is limitation of rib and diaphragmatic excursion, one will be able to obtain an explanation for the reduced function.[4] The upper respiratory tract and pharynx should be included in the fluoroscopic study.

The number of roentgenograms and the technique to be used should be decided after fluoroscopy. A minimum study should include roentgenograms in inspiration and expiration; in each oblique view (5 to 15 degrees); in each lateral view; and a posteroanterior roentgenogram with a Potter-Bucky diaphragm. It may be decided to do laminagraphic studies and rotational laminagraphy, selective angiography, bronchography, or some other study.

In approaching a comprehensive roentgen examination, one should keep in mind that the radiologist has an opportunity to make a real contribution. For example, although lung volumes are easily measured by the pulmonary physiologist, they vary so widely in healthy persons that figures obtained for pulmonary capacity do not necessarily reflect the presence of pulmonary insufficiency.[3] The most important aspect of pulmonary ventilation is the amount of air that reaches the alveoli and the uniformity with which it is distributed to them.[3] Any condition where air is prevented from reaching the alveoli in sufficient amounts results in hypoventilation with its consequences of anoxemia, CO_2 retention, and respiratory acidosis.[3] Conditions such as asthma, asbestosis, silicosis, other pneumoconioses and emphysema are only a few that produce hypoventilation.

Variations in alveolar ventilation/blood flow ratios may be diagnosed by exclusion in anoxemic patients. Hypoventilation can be eliminated by simple clinical tests (lung capacity, roentgenograms for anatomic changes). Impaired diffusion of O_2 across the alveolar capillary membrane can be measured by more elaborate physiologic tests (CO_2 uptake).[3]

Structural changes in the alveolar walls may produce anoxemia in the presence of normal alveolar ventilation and alveolar blood flow by imposing a barrier to the passage of O_2 across the alveolar-capillary membranes.[3] This occurs in individuals with pulmonary fibrosis, pulmonary emphysema, and other conditions such as some of the pneumoconioses; also mitral stenosis in an advanced stage. It can be detected clinically, by determining that arterial O_2 saturation falls during exercise and is restored to normal values during the inhalation of O_2.[3]

These are only a few examples illustrating an intriguing opportunity for radiologists to participate in a program of correlating radiologic data with physiologic studies. Barden and Comroe,[4] and Wright[85] have demonstrated the possibilities. It now behooves those of us who are located in institutions where there are good physiologic departments to do our part. In improving ourselves, it is not unlikely that real progress may be made in the detection of pulmonary dysfunction.

III. *Workers in an Industry-Dust Hazard Known or Unknown.* With the industrial group, a different program is necessary. It is essential that the employers of workers in any industry or process known to involve exposure to injurious dusts engage the services of a good clinician. He must be familiar with the plant operation and must know the workers—their occupation in the plant and their home environment. Such a physician is held in high esteem by the workers. This is important. Also the services of an industrial hygienist for periodic conventional dust counts are most helpful.

Whenever possible, the roentgenograms of employees should be made at the plant. This keeps the worker on the job, and one is more likely to obtain roentgenograms of good technical detail once a year. When the studies have to be made away from the plant, not infrequently serial studies will be interrupted for several years at a time.

Usually a good history is obtained, a

careful physical examination is made and the laboratory studies are limited to a test for syphilis, a blood cell count, a urine examination and a sedimentation rate determination.

The roentgen studies have varied over the years from stereoscopic and lateral exposures to single posteroanterior roentgenograms. At present I am planning to recommend a new program for new employees with the view of trying to test certain techniques for demonstrating changes in lung dynamics. It is hoped that the following studies can be made on all new employees:

1. Posteroanterior roentgenogram during inspiration. Posteroanterior roentgenogram during expiration. The tube level should remain the same.
2. Posteroanterior roentgenogram with expiration and inspiration recorded on the same film as a double exposure. The tube level should not change for the two exposures. The expiratory phase of respiration should be exposed first, otherwise one will not get good records of the two positions of the domes of the diaphragm and the ribs in inspiration and expiration.
3. Right lateral roentgenogram of the chest in inspiration and expiration. The tube should be kept at the same level.
4. For the succeeding three years, a single posteroanterior roentgenogram of the chest is to be recommended.
5. The fifth year studies will be the same as the pre-employment examination.
6. The full study will be repeated at five-year intervals thereafter, with single posteroanterior roentgenograms during the intervening years, unless some abnormality develops. It is hoped that such studies will compensate for the lack of opportunity to make careful fluoroscopic examinations.

As stated before, except for individual cases and a few collective studies on anthracite and bituminous coal miners, pottery workers, iron miners, foundry workers and asbestos workers, my experience has been limited to a study of a single industry in which the dust hazard is essentially pure quartz in a finely divided state. The workers are middle class Americans and the great majority are white people. This study has extended over twenty-two years.

In many of the individual cases, we have been able to obtain previous roentgen studies, making it possible therefore to learn something of the natural history of the disease.

For some years, Tuddenham,[80] one of my associates, has been working on basic concepts of visual physiology as it concerns the radiologist. His work has been helpful in explaining some of our deficiencies. Some of these include: the avoidance of outside major glare sources striking the retina; working in a room with low lights instead of complete darkness or bright illumination; and the use of diminishing lenses. The viewing room has 60 viewing boxes extending over one wall so that the roentgenograms of a complete case can be set up at one time. With double reading we think this plan reduces the number of errors in detecting abnormalities and changes from year to year. Looking at several roentgenograms, then removing those and replacing them with others of the same case, oftentimes leads to errors. With few exceptions, more than one radiologist studies the roentgenograms at the same time. This is most helpful in that it, on many occasions, prevents one from overlooking not only small but perfectly obvious variants or lesions. I have said repeatedly that if I were working by myself in a small community, my technician or helper of any category would be invited to participate in my studies at the time of reporting. Every radiologist has had or will have a member of his nonprofessional staff call to his attention a shadow that was not evaluated in a report that has been prepared to be sent to the referring physician.

SIMPLE SILICOSIS

The characteristic lesion in silicosis is a

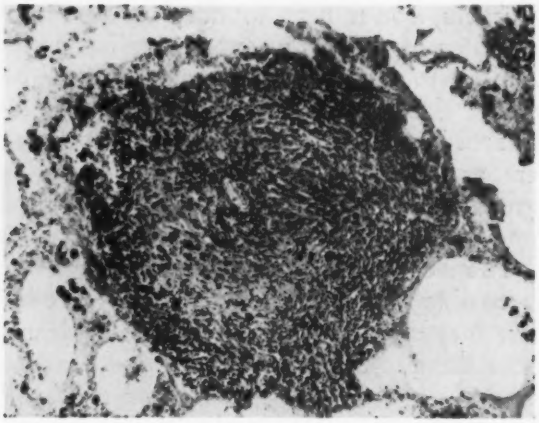


Fig. 1. Early silicotic nodule showing the collection of epithelioid cells which look like the tubercle formation of tuberculosis. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

circumscribed nodule of hyaline fibrosis (Fig. 1 and 2). The earliest lesions are invisible or are recognizable only microscopically or with a magnifying glass.[24] These parenchymatous nodules may continue to develop and ultimately reach a size of 3 to 4 mm. in diameter. Pathologically, the nodules have well-defined borders except when there are accumulations of nonsiliceous dusts.

The earliest roentgen findings that I accept as evidence of simple silicosis are the small, multiple shadows, 2 to 6 mm. in diameter, more or less uniform in density, which do not disappear in a roentgenogram made with slight rotation. Shadows that disappear with slight rotation are likely to be vascular (Fig. 3, 4 and 5). The blood vessel shadows are denser and their borders are more sharply defined than are those of nodular shadows. The shadows of the silicotic nodules are usually distributed along and between the vascular channels and the bronchial tree of both lungs, and, on the roentgenogram, at times, they may be limited largely to one lobe. Not frequently, even though a bilateral distribution of the shadows occurs, does one see them in the apical, peripheral and lower portions of lung fields.

The roentgen appearance of the shadows

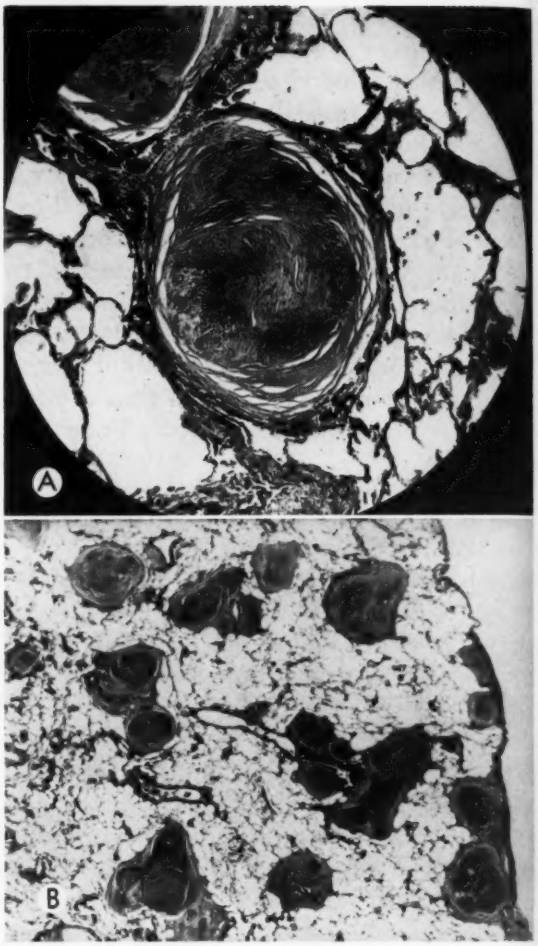


Fig. 2. (*A*) Silicotic nodules showing the hyalin collagen fibers. Note that these nodules are often adjacent to each other. The nodules have varying shapes and are not cylinders of tissue; consequently, they do not appear as well-defined circular shadows on the roentgenogram. They do not produce characteristic roentgen shadows that allow one to differentiate such shadows from the shadows of other conditions such as siderosis or miliary tuberculosis solely by the roentgen examination. (*B*) Larger field of the same case as *A*. Note the varying sizes of the nodules and their distribution not only in the lung parenchyma, but also along the visceral pleura. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

of the silicotic nodules is not characteristic and not as diagnostic as one would wish (Fig. 6, 7 and 8). Some are round, some oval, some irregular, some large and some small. Most of the lesions have a uniform density, but some have shadow densities in

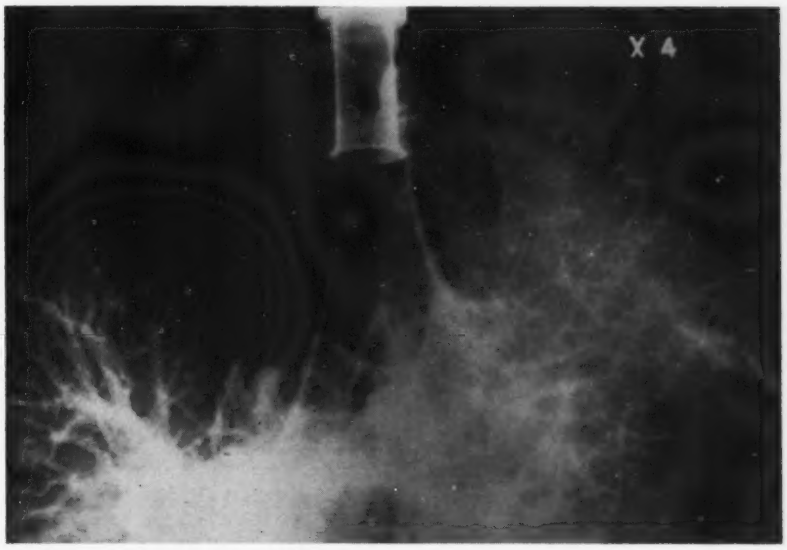


Fig. 3. Roentgenogram of excised and inflated lungs. The pulmonary vessels of the right lung have been ligated; those of the left lung have not, thus they contain less blood. Note the prominence of the vascular shadows of the right lung as compared to those of the left. The vascular system when filled with blood, for the most part, produces the roentgen pattern that is called truncal markings. Normally, these truncal markings vary in size and prominence with the cardiac cycle, the phase of respiration, and the shunt mechanism.

their centers simulating that produced by calcium. The shadows of these lesions are called *nodulation*, which unwittingly implies that they can be distinguished from mottling due to tuberculosis or some other infectious process. I have frequently found that the periphery of the silicotic nodular shadow is so ill defined that in itself it can-

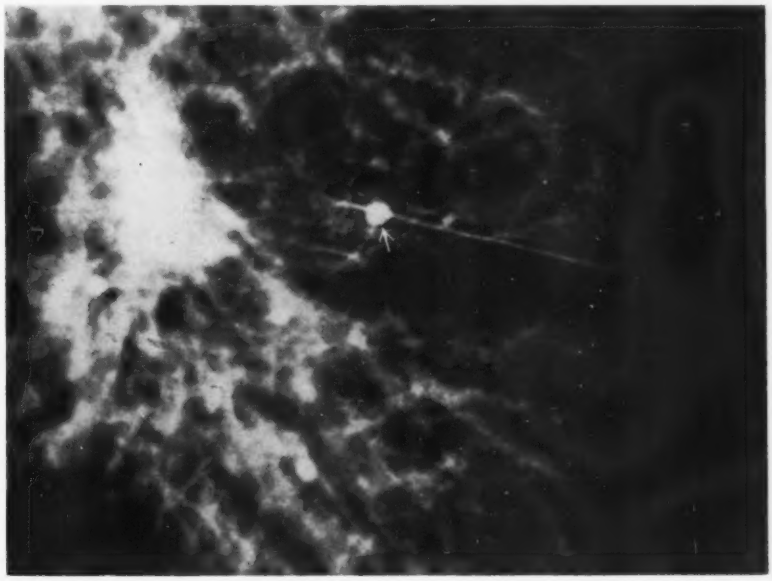


Fig. 4. Roentgenogram of excised and inflated lungs showing the appearance of well-defined circular and dense shadows of blood vessels in an axial view. The dense shadows at the site of the arrows are blood vessels as determined by careful anatomic dissection.

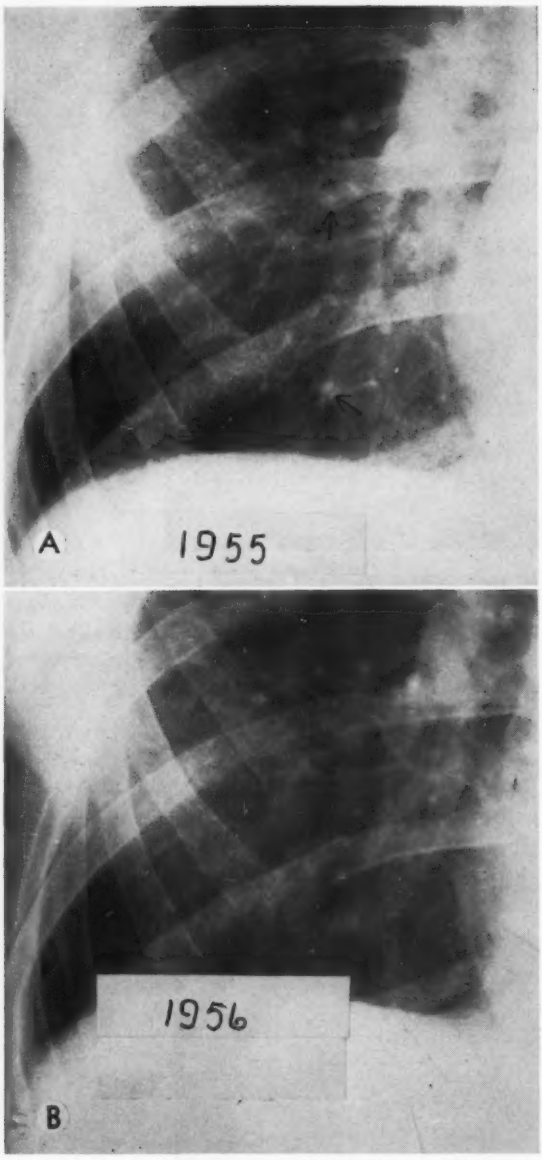


Fig. 5. (*A*) Roentgenogram showing the shadows of blood vessels in a forty-eight year old individual who works in a silica industry. The blood vessels appear quite dense in the axial view and should be differentiated from silicotic nodulation. This examination was made in 1955. The blood vessel shadows in this instance are almost as dense as those produced by calcium. (*B*) Roentgenogram made one year later. Note that the blood vessel shadows in the right lower lobe in the 1956 examination are not quite so prominent as those seen in 1955. It is interpreted that in the 1955 examination the vessels were filled with blood, and one sees their shadows in the axial view much more prominently than when they are not filled with blood.

not always be differentiated from mottling in miliary and other types of tuberculosis and small shadows due to a generalized metastatic malignancy. In most instances, however, a correct diagnosis can usually be made by correlating the roentgen findings with the history and clinical data.

I have restricted myself in the diagnosis of nodulation to those shadows which are apparent enough, so that in the majority of instances a diagnosis of nodulation is likely to be made the following day or the following week (Fig. 6). Even then, my experience has shown that on occasions, a diagnosis of nodulation is made; while the following year, after again reviewing all of the roentgenograms and comparing them with the new study, one finds it difficult to be sure about the presence of nodulation. On other occasions, the opposite obtains—that is, one hesitates to make a diagnosis of early nodulation after comparison with the succeeding examination.

In my experience, if one anticipates too much by trying to make a very early diagnosis and thereby calls every little shadow (1 to 3 mm.) evidence of pneumoconiosis, errors are committed not infrequently. In interpretation, one has to contend with the competition of confusing shadows from film grain size and vascular movement, blood vessel shadows, and shadows of superimposed soft tissues, all of which are present on every roentgenogram. Overinterpretation, for the most part, is a common fault in medical practice and in radiology in particular. The desire is to be meticulous and not overlook early manifestations of disease so as to protect the patient. The big areas where overinterpretation of chest roentgenograms is practiced are early carcinoma of the lung, pneumoconiosis, and possibly tuberculosis. One questions the value of such overinterpretation, especially as it concerns pneumoconiosis.

One of the interesting questions that arises regarding the silicotic patients as seen by the radiologist is that some fail to exhibit generalized nodulation that can be

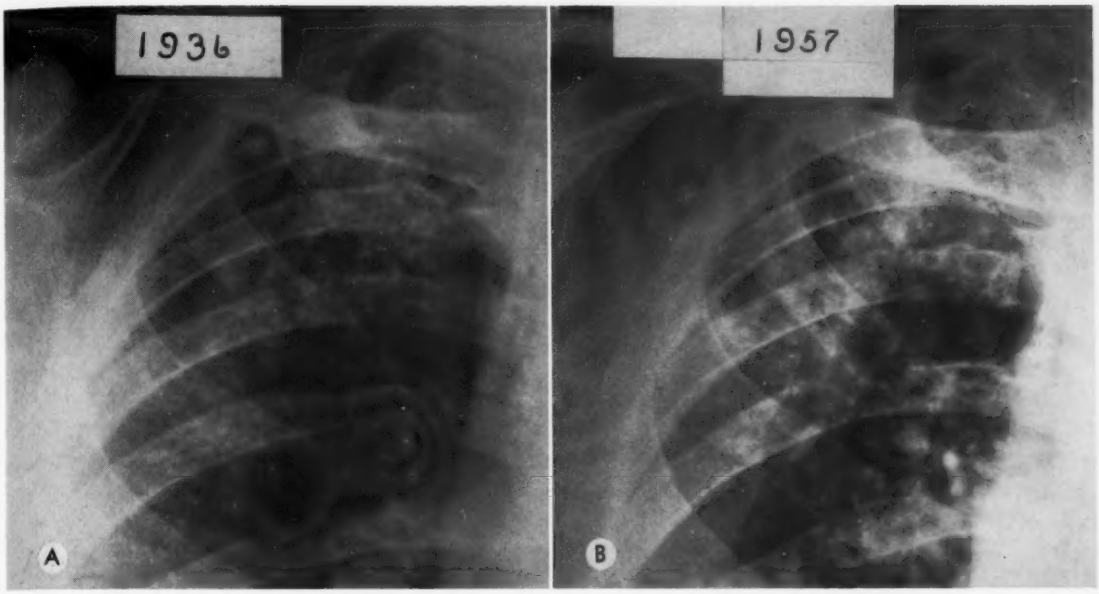


Fig. 6. (*A*) Early silicotic nodulation in a pure quartz worker in 1936. (*B*) Conglomerate silicotic lesion in the same individual twenty-one years later. This worker is asymptomatic and has been so for several years, so his lesion probably falls in the category of simple silicosis.

seen on the roentgenogram. This observation apparently is true even when men are working under similar conditions in the same industry, and, up to the present time, there is no adequate explanation.

When nodulation occurs, the nodules may or may not show progressive changes. In a few cases that have been followed for fifteen years or longer, I have seen the pattern of nodulation replaced by massive shadows, with either complete or almost complete disappearance of visible nodular shadows on the roentgenogram.

One may or may not be able to demonstrate a definite enlargement of the hilar lymph nodes.

Another lesion generally included under the classification of simple silicosis is the small *conglomerate lesion* (1 to 5 cm. in diameter). Such lesions may result from a combination or coalescence of discrete nodules, or the lesion, primarily, may occur as such.

Conglomerate lesions are usually localized and do not occur in the same portion of the lung in every patient. On the other hand, roentgenographic studies of patients with such lesions frequently show them in the upper half of the lung fields (Fig. 6). Microscopic examination of the tissue from such areas often reveals no evidence of infection (Fig. 9). The nodules seem to be closer together than in other portions of the lung; they are less uniform in size, and they are embedded in a matrix of diffuse, fibrous tissue having the same characteristic appearance as that forming the nodules themselves.[25]

The shadow of the conglomerate lesion in simple silicosis is often difficult to distinguish from that found in silicosis with infection. From the roentgenologic standpoint it is impossible to differentiate in a single examination, whereas serial examinations may or may not show slight changes in the extent of the shadows of lesions complicated by an active infection. Roentgenograms made with a Potter-Bucky diaphragm and/or laminagrams made in the erect posture provide additional information which is helpful in evaluating the presence or absence of an infection.

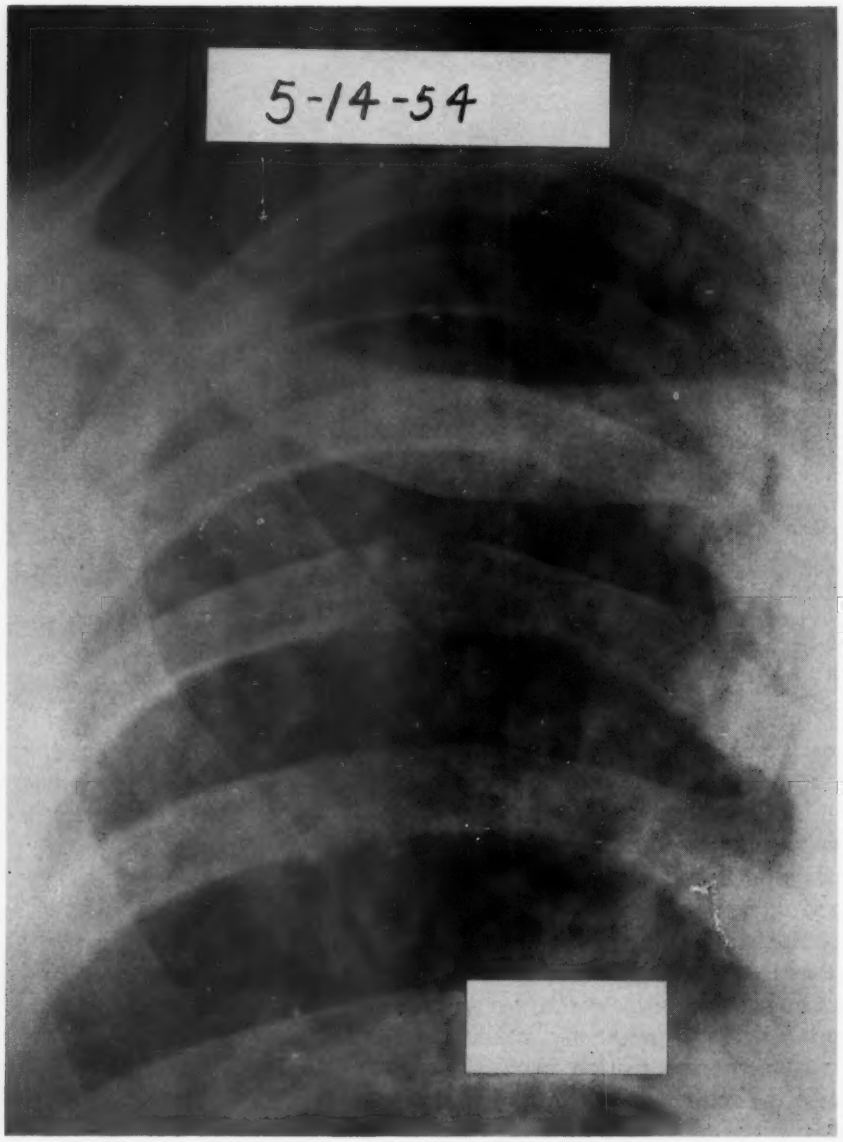


Fig. 7. Sarcoidosis of the lung in an individual twenty-six years of age. Note the pattern of the lesion in the right upper lobe. It could be mistaken for a pattern of a silicotic process. This case was proved by a scalene biopsy.

Including the conglomerate lesion under the category of simple silicosis is only justifiable in those instances when repeated examinations over several years fail to show any roentgen evidence of change. The conglomerate lesion in simple silicosis is not a thick dense lesion, but irregular, thin, having its longest diameter in the vertical position. If, after all of the above qualifications have been considered, there is still doubt as to the true nature of the lesion, my plan is to regard the lesion as one of silicosis with infection either quiescent or chronic.

The presence of well developed emphysema may or may not be observed in simple silicosis. One cannot be certain whether the emphysema is due to silicosis or not, especially if the pattern of nodulation is slight. When well advanced, it is relatively easy to diagnose with either the fluoroscop-

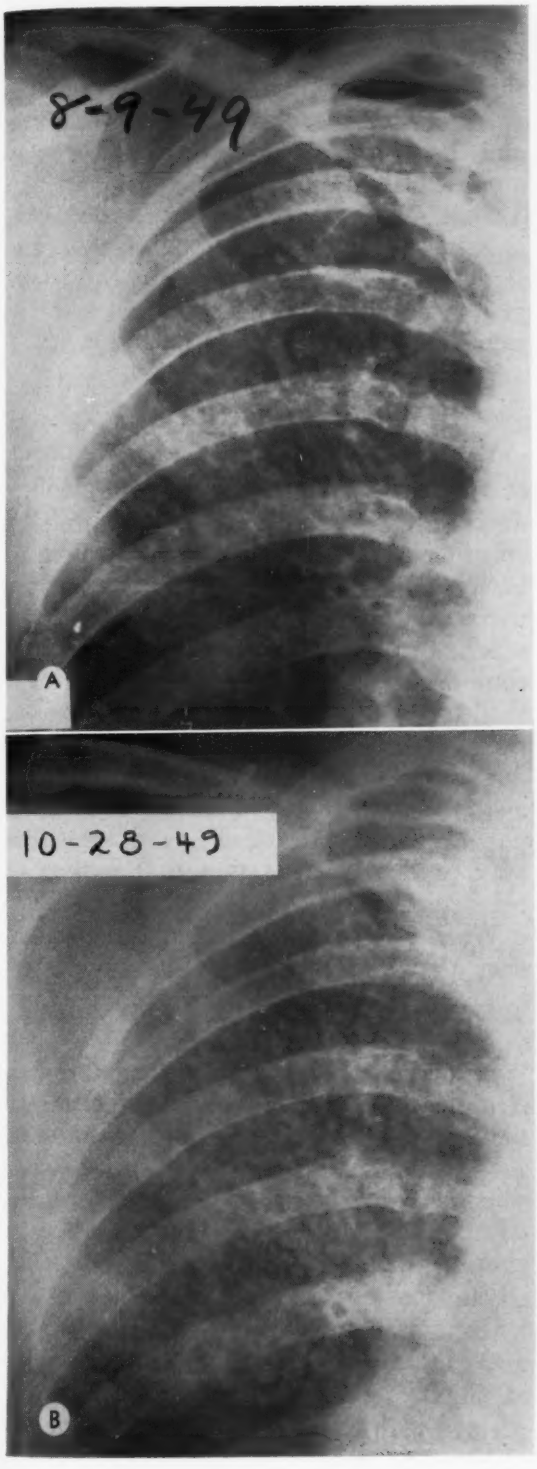


Fig. 8. (*A*) Hemosiderosis in a man, thirty-seven. On August 9, 1949, there was evidence of a pattern of nodulation in the upper lobes of both lungs, the right being shown here. (*B*) This examination was made two months later. Note the tremendous increase in the pattern of nodulation. Shortly after this, the individual was operated on and following a commissurotomy, died from a pulmonary embolus. Microscopic studies showed the presence of hemosiderosis.

ic or roentgenographic examination. The barrel chest; the restricted movement and flattening of the domes of the diaphragm; the increased size of the preaortic space; the increased volume of the lungs seen in the heavily exposed roentgenograms made in the erect and horizontal positions; the loss of fine lung detail in conventional exposures; the widening of the interspaces; and the demonstration of bullae in supervoltage roentgenograms are some of the criteria for diagnosis.

In focal emphysema of slight to moderate degree, careful fluoroscopy with well accommodated eyes sometimes gives one the best lead. Increased pulsation of the pulmonary artery may be present. Overexposed conventional and kyphotic roentgenograms are helpful. In many instances, we believe that emphysema can be demonstrated by supervoltage roentgenograms

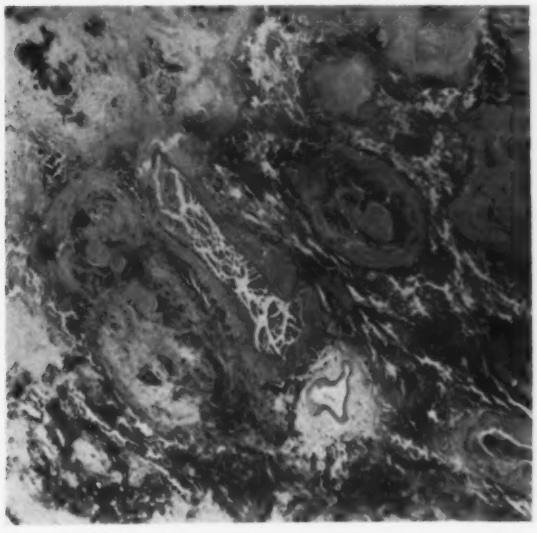


Fig. 9. Modified silicosis in a conglomerate mass from a hematite miner. One does not see the well-defined silicotic nodules, such as shown in Figure 2. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

better than by other roentgen procedures.

Enlargement of the hili is very difficult to demonstrate with certainty. Overexposed or supervoltage roentgenograms in the posteroanterior, oblique and lateral views are necessary. Laminagrams and rotational roentgenograms are helpful. Comparison with previous roentgenograms provides the most reliable information if the roentgenograms are exposed well enough to differentiate between vascular and lymph node shadows.

SILICOSIS WITH INFECTION

In this group are included all cases of silicosis with detectable evidence of infection. It is not always possible in the living patient to determine whether the infection is active or inactive even in instances when conventional clinical and laboratory examinations are available. Under such circumstances, mistakes will occur, but if one exercises good judgment, the affected person can be protected by taking the necessary precautions.

The lesions (either some or all) described as occurring in simple silicosis may be modified by infection. Other lesions that may be found include: cavities (usually thick walled)—tuberculous in origin; lesions occurring as a result of anemic infarcts; massive lesions; mottling; soft nodulation; distortions of the tracheobronchial structures; bronchiectasis; Reichmann's "rainstorm" streaking, distorted vascular pattern; mediastinal displacement; extensive calcification in the hili, paratracheal region and in the lung fields; various degrees of emphysema and bleb formation; pleural thickening, interlobar and peripheral; pleural collections; pneumothoraces; and deformations (flattening, peaking, individualization of costal components and adhesions) of the domes of the diaphragm. The radiologist is rarely able to predict whether the infection is due to the tubercle bacillus or some other organism. One suspects, however, that in the majority of instances the superimposed infection is tuberculosis, for, as stated by Gardner,[25] the postmortem examinations showed an element of tuberculosis in 60 per cent of the cases studied by him. Other infections and infestations include: staphylococcic and Friedländer's pneumonia; histoplasmosis; and fungus diseases.

Some of the detailed changes occurring in the respiratory and cardiovascular structures as demonstrated by the roentgen examination in silicosis with infection[16,28,63–67,70] are summarized below.

TRACHEA

The tracheal shadow may be in a normal position, especially if a tuberculous process or some other infection is superimposed upon an already established silicotic process. In other instances the trachea is displaced backward and/or to one side. Frequently, there are calcified lymph nodes in the paratracheal region. They may be irregularly or completely calcified. Some have an eggshell type of calcification (Fig. 14). At one time, we thought calcification was largely due to tuberculosis. Now we know that histoplasmosis, coccidioidomycosis and possibly other infestations and infections may produce calcification that looks like tuberculosis.

When calcification is incomplete, we suspect that the process is not completely healed.

HEART AND AORTA

On occasions, evidence of cor pulmonale is observed in the advanced silicotic patient. Many clinicians[16] and others have regarded this condition as a complication of silicosis. The relationship between cause and effect is difficult to establish in this instance, and some cardiologists who have given considerable thought to the question find themselves unable to express an opinion. Many of us who have been interested in silicosis for years have not been impressed with the incidence of cardiac complications. On the other hand, it is possible that with increased use of modern cardiac studies in the well-advanced silicotic patient, more evidence of heart changes will be found. But even if such evidence is

found, the cardiologist and the general pathologist know that in the older age group of men not exposed to harmful dust, who are studied at autopsy, cardiac complications are not uncommon.

DOMES OF THE DIAPHRAGM

If considerable emphysema is present, the domes of the diaphragm may be depressed and limited in their movements. At times it is necessary to have the patient cough or sniff in order to demonstrate, roentgenoscopically, evidence of diaphragmatic excursion.

Another abnormal appearance is multiple peaking of the domes of the diaphragm. It may be impossible to differentiate the peaking caused by pleural adhesions from that due to inelasticity of certain structures of the lung. Both causes may be present. Many bizarre appearances of the domes result from emphysema and contractions secondary to lesions within the lung and pleura.

HILI

The shadows of the hili may or may not be within normal limits. They may be enlarged or may be partly or totally obscured by larger shadows produced by lung lesions. Each hilus may or may not be displaced upward, laterally or backward by contracting scars of infected silicotic processes in the lungs. The roentgen appearance of the hili may present a rather homogeneous shadow as contrasted to the normal. There may be calcified lymph nodes in one or both hili. The lymph nodes either may be densely calcified, irregularly calcified, or the calcium may be deposited in such a way as to produce an egg-shell appearance. Calcification of lymph nodes is regarded as evidence of tuberculosis, histoplasmosis or some other process. If the lymph nodes are not completely calcified, they are thought to be the seat of an infection that is not completely healed. Such evidence is not, as a rule, thought to be significant enough to justify recommending that the individual be removed from his particular occupation unless there are other modifying influences.

On the posteroanterior roentgenogram, one not infrequently sees a large shadow which may be unilateral or bilateral. For years these shadows were thought to be due to enlarged lymph nodes, and in some instances were treated as lymphoblastoma. Now we realize that they may represent a lung lesion (massive) which has migrated to the midline and its shadow is superimposed on the hilus, thereby posing a diagnostic problem unless one has previous roentgenograms which demonstrate the natural course of the lesion.[68]

LUNGS

The lung changes vary considerably. The lesions include: nodulation, soft nodulation, conglomerate and massive lesions, emphysema, emphysematous blebs, bronchiectasis, abscesses and cavities.

The term "linear markings" is not employed by us. I presume that those who recommend the term use it to describe shadows in the peripheral portion of the lung and not for the shadows of the vascular markings. Such shadows are so ill defined that we prefer not to classify them.

Nodulation. The shadows of nodulation are similar to those described under simple silicosis and cannot be demonstrated by a roentgen examination in every case of modified silicosis with infection. This is particularly true in certain industries such as hard coal mining and granite cutting. As already stated, the roentgen evidence of the pattern of nodulation disappears in some instances when massive lesions develop.

Soft Nodulation. The shadows of soft nodulation are much larger than those produced by nodulation and mottling, but are smaller than the conglomerate shadows. The description of such roentgenologic shadows is provided in order to emphasize a perinodular cellular reaction observed by the pathologist.

Massive Shadows. These shadows vary from 5 to 20 cm. in size. Some are round, some oval and some wedge-shaped. Some

involve an entire lobe or more than one lobe (Fig. 10). These lesions are usually due to extensive areas of fibrosis and are found anywhere. Frequently, they occur adjacent to interlobar fissures and involve either the basal portion of the upper lobes or the apical portions of the lower lobes or the process may extend across the interlobar fissure and involve both lobes. In lateral views of the chest, lesions are often seen lying posteriorly. Rotational laminagrams are helpful in demonstrating the relations of such lesions. With overexposed roentgenograms, laminagraphic studies or roentgenograms made with the Potter-Bucky diaphragm, it is possible to show some of the details of a massive lesion, such as distorted and dilated bronchi, emphysematous changes, effect on the vascular structures, cavities and areas of calcification and caseation.

At one time most of the massive lesions were thought to be due to silicosis with infection, but Riddell,[74] Gardner,[25] McCloskey[55] and others question whether this is always true. Besides infection and vascular occlusion, Gardner[25] indicated that a "third essential factor may be minerals other than free silica."

On conventional studies, air shadows in massive lesions simulate the appearance of cavities, but with laminagraphic examination in the erect posture, dilated major bronchi sometimes are found to be the cause of the air shadows (Fig. 11*A* and 11, *B–D*).

In conventional roentgenograms and laminagrams, at times, one sees shadows extending downward more or less perpendicularly from the massive lesions toward the domes of the diaphragm. These are blood vessels and have been called Reichmann's "rainstorm" streaks (Fig. 12). These vessels are particularly striking when filled by an opaque material (pulmonary angiography). The position of the vessels is due to the contraction of the lung structures produced by the massive lesion.

With selective angiography, one can now

Fig. 10. (*A*) Anthracosilicosis in a fifty-seven year old man. The massive lesions involve the upper portion of the lower lobes and they seem to have extended across the interlobar fissures into the lower portion of the upper lobes. This individual had shortness of breath, emphysema and cough. The costal expansion and contraction was good and there was no delay on inspiration and expiration. The expiratory movement of the domes of the diaphragm was quite prolonged (over ten seconds). (*B*) The lateral roentgenogram shows the position of the massive lesions. These lesions lie posteriorly.

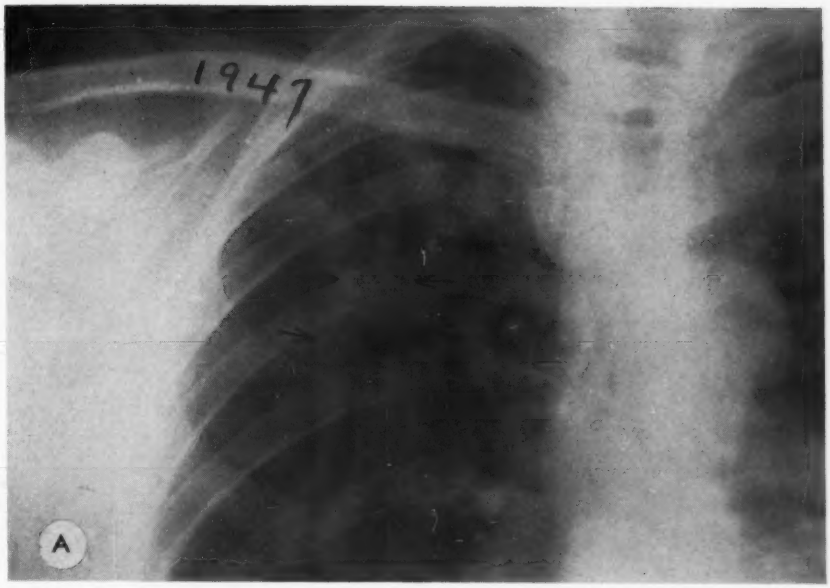
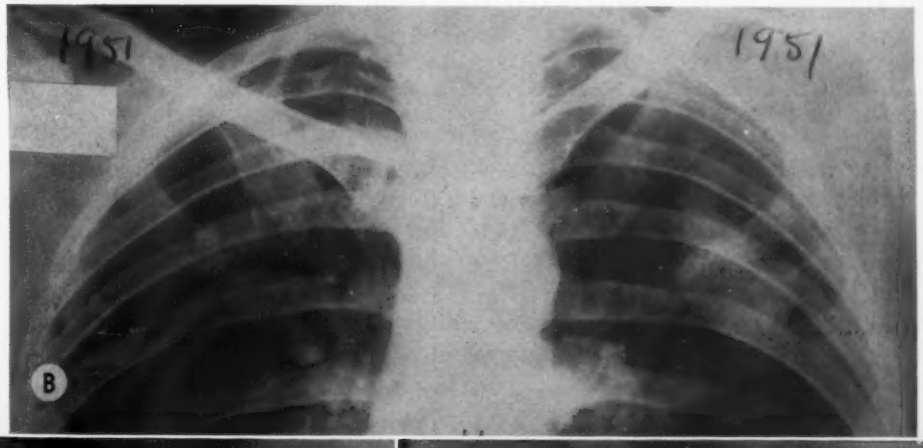


FIG. 11. (*A*) Conglomerate mass in the right upper lobe (arrows) in a young man who worked in an industry which had a pure silica dust hazard. In 1945, the chest was normal. In 1947, several months before this study, he had acute pneumonia. The roentgen manifestations of silicosis developed very rapidly thereafter.



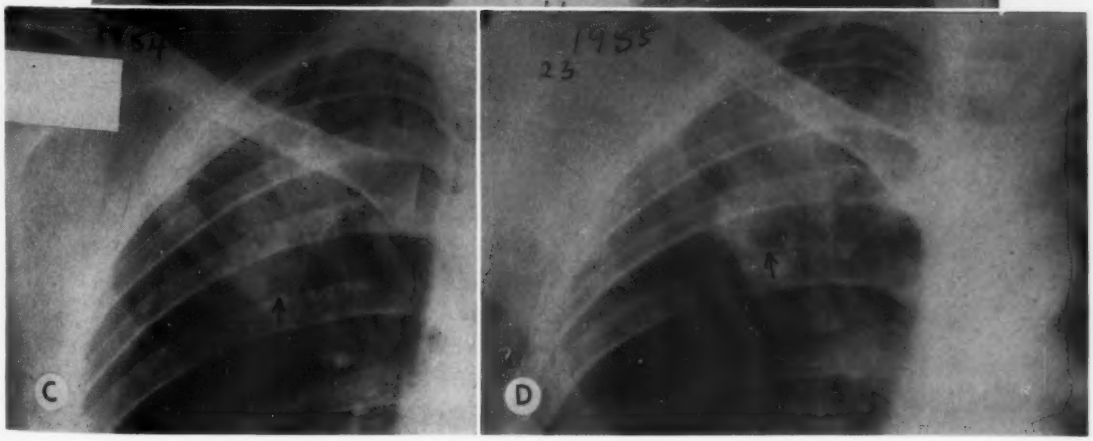


FIG. 11. (*B*) Roentgenogram made in 1951, four years later. There are conglomerate masses in each upper lobe. (*C*) March, 1954. A small cavity is developing in the lower portion of the conglomerate mass (arrow). (*D*) April, 1955. The cavity is larger. No tubercle bacilli have been demonstrated. A presumptive diagnosis of an anemic infarct has been made, but frequent examinations for tubercle bacilli are continuing.

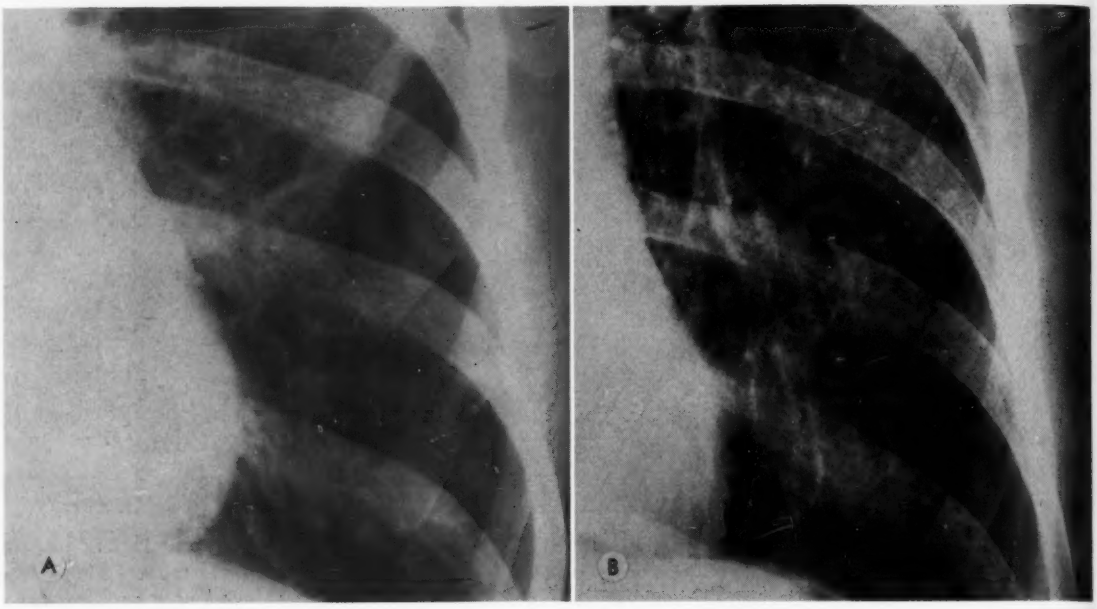


FIG. 12. (*A*) The appearance of the blood vessels as seen in 1936 in a person thirty-eight years old. The blood vessels in the left lower lobe are ill-defined. Compare these with *B*. (*B*) Note the stringy shadows of the blood vessels seen in the left lower lobe. This occurs after the contraction of the left hilus upward by the fibrosing lesion in the left upper lobe and produces what has been described as Reichmann's "rainstorm" streaks. These shadows are cast by the blood vessels. Same case as *A* which is regarded as tuberculosilicosis, involving each upper lobe.

demonstrate in the living patient, the fibrosing effect of the massive lesion on the surrounding blood vessels. Some vessels are completely obliterated; in others, the lumen is variously encroached upon (Fig. 13). Vorwald[81] tells me that in those patients with partial encroachment upon the lumen, but not complete obliteration, dyspnea is more dominant. This is a very interesting observation. It may be a corollary to that of the partial encroachment upon the lumen of the renal artery (Goldblatt's kidney); *i.e.*, where one may find hypertension with partial, but no elevation of blood pressure with complete obliteration.

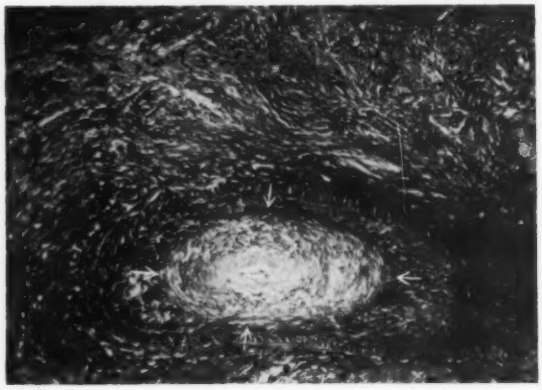


FIG. 13. Endarteritis obliterans occurring in a silicotic person with a conglomerate mass which was an anemic infarct with liquefaction necrosis. (Courtesy of Dr. Arthur J. Vorwald, Wayne University, Detroit, Michigan.)

Selective angiography will show displacement of vessels in patients who have emphysematous blebs and bullae, and in localized pneumothoraces and pleural collections.[86]

Calcification in massive lesions is regarded as evidence of tuberculosis or histoplasmosis (Fig. 14). Sputum examinations and skin tests may be helpful in making the diagnosis.

We have observed a migration of massive lesions toward the hilus in certain instances (Fig. 15).[68] Just why this observation was not recorded years ago may be explained on the paucity of serial examinations and a failure to put all of the roentgenograms on

Fig. 14. Tuberculosilicosis or histoplasmosilicosis in a sixty-seven year old individual who has been followed for over twenty years. The calcification in the massive lesion has gradually increased over the years. This roentgenogram shows some of the types of calcification that may occur in histoplasmosis or tuberculosis.

the view boxes at one time. In other words, if one looks at the roentgenograms of serial studies one at a time, the slow migration will be missed. In some of the workers with lesions near the midline, in our first study, it was possible to get earlier roentgenograms which when compared with ours provided a clue as to what was happening.

After noticing the migration of the massive lesions, I studied the work of Lubert and Krause on lobar collapse and bronchopulmonary segments,[46,48] and learned that they were observing migration in lung lesions. These authors[48] state: "The lobes do not merely shrink to become smaller replicas of themselves. As they become smaller they change from a three-dimensional, pyramidal or conical figure to approach a two-dimensional triangle and flatten along the mediastinal and parietal pleura. With continuation of the collapsing force, they move along the curved surface of the chest wall toward the hilus, the direction of the movement depending on the natural configuration and position of the lobe and the presence or absence of modifying factors." The observation of migration has been helpful, and explains the pathologic physiology simulating the shadows of greatly enlarged hilar lymph nodes.

Kirby[45] has operated on and removed 9 massive lesions, the operations being performed because of a diagnosis of suspected carcinoma of the lung. The lesions were unilateral single masses usually found in hard coal miners, without a pattern of nodulation (Fig. 16 and 17). A similar appearance was seen in quartz workers. All of the lesions proved to be silicotic, without evidence of carcinoma.

The roentgen diagnosis in the first of Kirby's patients was "probable carcinoma." On that basis, the patient had a lobectomy. I was upset over the roentgen diagnosis because the lesion had the appearance of a silicotic massive lesion. When the second patient came along, I was asked to give an opinion. On the basis of a single lesion and marked encroachment on the lower lobe bronchus, a diagnosis of carcinoma was made. At operation, the lesion

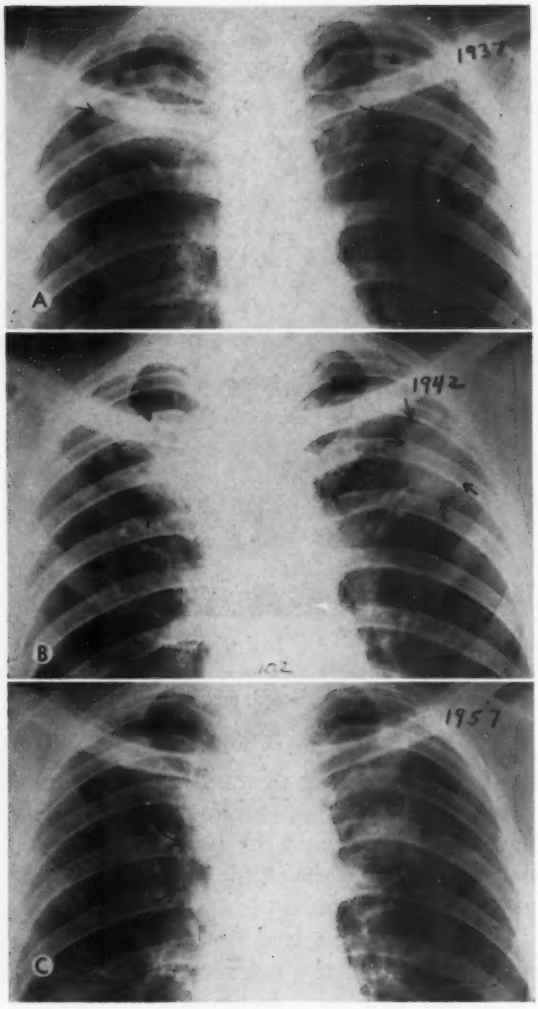


Fig. 15. (*A*) Silicosis with infection in an individual twenty-seven years of age. This examination was made in 1937. Note the lesions in both upper lobes as indicated by arrows. (*B*) Silicosis with infection in a thirty year old individual who was exposed to essentially pure quartz. Note the lesion in the left upper lobe which is close to the periphery. The lesion in the right upper lobe has migrated toward the mediastinum. This examination was made in 1942. (*C*) Roentgenogram taken in 1957. The lesions in the upper lobes are continuing to migrate toward the hilus. The one on the right side is scarcely seen at this time. The one on the left side has migrated considerably since the previous examination.

proved to be a silicotic mass. Approximately one year later, the follow-up roentgen study of the chest showed a large mass in the upper lobe. It was thought that a carcinoma was missed at the first operation so the patient was re-operated upon. A silicotic mass was found at the second operation. A recent examination, seven years after operation, showed no massive lesions.

The development of lung masses following operation is thought to be an aftermath of lung trauma. Because of that observation, I have been somewhat concerned about lung biopsy. The experience of Theodos *et al.*[79] and Effler and his collaborators[17] in over 100 individuals who had lung biopsies done under local or general anesthesia shows that the procedure is safe and quick and that an accurate diagnosis can be obtained.

If Vorwald is correct in his impression that massive lesions with partially occluded vessels may be a dominant factor in causing dyspnea, one should consider removal of such lesions if by selective angiography one can demonstrate evidence of partial occlusion of the vessels.

We are now fully aware that nodulation is not necessary in order to make a diagnosis of silicosis in an unilateral massive lesion in a coal miner. The thoracic surgeons are not as convinced of that fact as we are at this time. Krause and Lubert[46] made an observation which should be considered when making a diagnosis of carcinoma. They feel that the presence of a shadow of unknown etiology in an anterior segment should impart a feeling of special urgency because in these instances the possibility of neoplastic disease is somewhat greater. Although many of the massive silicotic lesions are posterior, we have found them in the anterior segment of the upper and lower lobes.

Emphysema. Emphysema may be focal or generalized. Gough,[31] Fletcher,[19] Gilson and Hugh-Jones,[27] and other English investigators,[10,11,22,32,50,51,53,78] in excellent studies, have re-emphasized the difficulties in determining the presence or absence of emphysema or its extent, when present in coal miners. We will consider emphysema under another heading.

In pure quartz workers emphysema is present, but is not the major factor that one

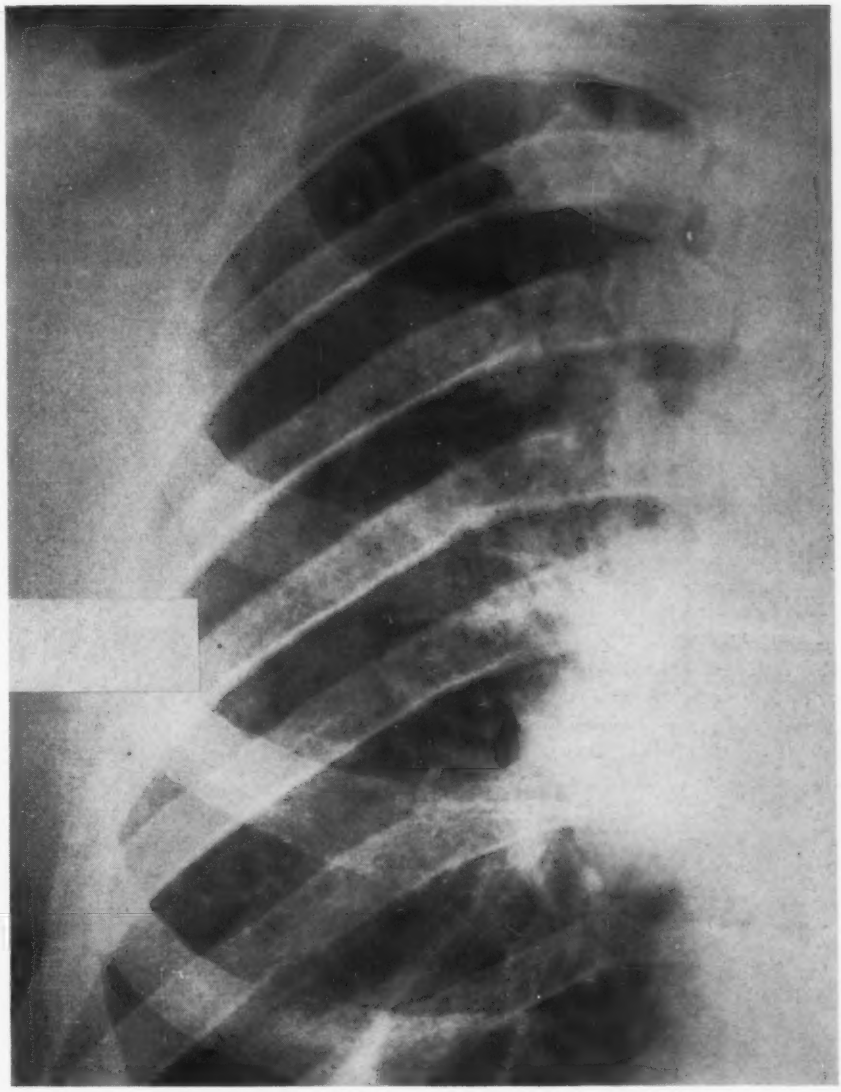


Fig. 16. Anthracosilicosis in a fifty-seven year old coal miner simulating a carcinoma of the lung. This individual had a twelve year history of cough and dyspnea. There was a 22 pound weight loss in two years. Bronchoscopy had revealed an extensive obstruction of the right main bronchus, with blood stained secretions. The Papanicolaou smear was regarded as negative. A roentgen examination in November of 1948 showed a mass at the right hilus. There was no roentgen evidence of nodulation. Laminagrams of the right hilus area showed evidence of encroachment on the lumen of the right main bronchus. The patient was operated on and the mass was found to be anthracosilicosis.

sees in the coal miner. Gough[31] expressed more concern as to the effect of emphysema on the heart than one usually hears about in this country. Dyson,[16] in his comments about the heart, was talking largely about his experience with coal miners.

When focal emphysema is present, its diagnosis is not easy, as mentioned previously. The supervoltage roentgenogram is sometimes helpful in showing focal emphysema along the peripheral portions of the lung.

Bleb and Bullae Formations. These can readily be seen in most instances. If very large, they will displace and distort the vascular and bronchial trunks.

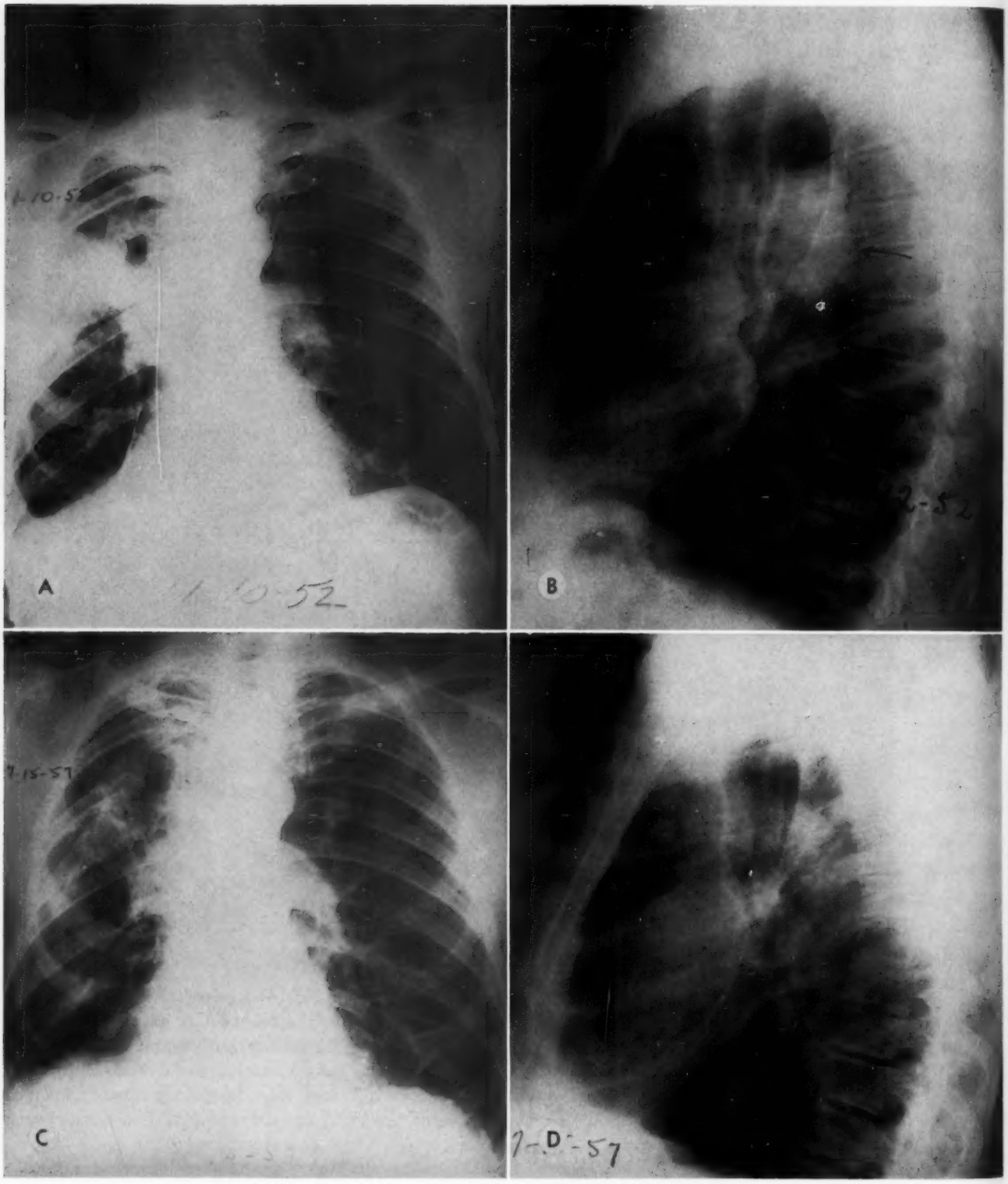


Fig. 17. (*A*) Same case as Figure 16, four years later (November 10, 1952). There is a mass in the right upper lung field which from the roentgen standpoint was interpreted as anthracosilicosis. The surgeons thought it was a carcinoma. (*B*) Lateral roentgenogram, showing the position of the mass. The lesion was removed and found to be an anthracosilicotic mass. (*C*) This roentgenogram was made in July, 1957. There has been no return of the anthracosilicotic mass. The shadow in the right upper lung field is due to the appearance of the ribs following thoracotomy. There is no roentgen evidence of anthracosilicosis at this time. (*D*) Lateral roentgenogram made in July, 1957.

Bronchiectasis. This, when present, may be demonstrated by erect and horizontal laminagraphy or by the injection of a water-soluble opaque medium. The bronchiectasis may or may not be due to the silicotic process. If there is no causal relationship, the bronchiectasis is often observed in the left lower lobe, but it may be present anywhere.

Abscesses. Following pneumonia or metastasis from infected lesions elsewhere, abscesses, when complicating a silicotic process, act very much like abscesses in nonsilicotic lungs, except that they may be resistant to antibiotic therapy. If the inside of the abscess wall undergoes epithelization, it becomes chronic with intermittent inflammatory reactivation. When this occurred, lobectomy or some other surgical procedure had been performed in our cases.

Cavities. These are most often due to a tuberculous process, but are also seen in carcinoma. The walls may be thin or thick.

Where the silicotic process is quiescent, the cavity may be thin walled as is seen in tuberculosis without a complicating silicosis. When the silicotic process is active, the wall is often thick, irregular and shaggy. I have not had enough experience with the use of antibiotic therapy in these cases to have an opinion as to their response to treatment.

PLEURA

Occasionally, one sees a pneumothorax complicating silicosis. In such instances the extent of any pleural thickening may be studied. Empyema and other pleural lesions may occur. Since the onset of antibiotics and sulfonamide therapy of lung infections, one rarely sees empyema in the silicotic patient.

TUBERCULOSILICOSIS AND SILICOSIS WITH TUBERCULOSIS

The roentgen manifestations of tuberculosilicosis are protean and yet distinctive.[70]

Only rarely is one fortunate to see the evolution of the entire process in a given patient. It would seem, however, that the pathologic evidence is sufficient and that adequate numbers of cases have now been followed by means of serial roentgenograms over a period of years to warrant a presumptive diagnosis of tuberculosilicosis[5] either upon the visualization of nodulation with concentration, coalescence or conglomeration, or, when the involvement is bilateral, upon the demonstration by themselves of large areas of conglomerate or massive fibrosis. Commonly, these areas of conglomerate fibrosis continue over the years to increase slowly in size, incorporating within themselves more and more silicotic nodules from adjacent portions of the lungs, until finally one may have as the end-result either single or multiple, unilateral or bilateral massive shadows of fibrosis (Fig. 18). As healing takes place, calcification often is observed in heavily exposed roentgenograms. The trachea, hili, and other mediastinal and lung structures may be displaced by scar contraction, especially if the lung lesion extends to and involves the pleura. One may see evidence of varying degrees of emphysema. The changes may be so extensive as to obscure the details that are associated with either tuberculosis or silicosis. As these individuals are not toxic, it is thought that the emphysema accounts for the disability, but disturbed blood supply may also be a factor.

As indicated previously, some of these lesions undergo cavity formation. It should be borne in mind that histoplasmosis may simulate a tuberculous process so far as calcification is concerned. We have observed extensive changes take place over a period of three to five years in some patients who were diagnosed as having tuberculosilicosis, but in whom no recognizable tuberculosis was found at the postmortem examination. Limited reservation must, of necessity, be entertained, however, until the case for or against nontuberculous infection as the etiologic agent is definitely proved.

Silicosis with tuberculosis may occur in either of *two* forms, the clinical course and roentgen behavior of which are as distinc-

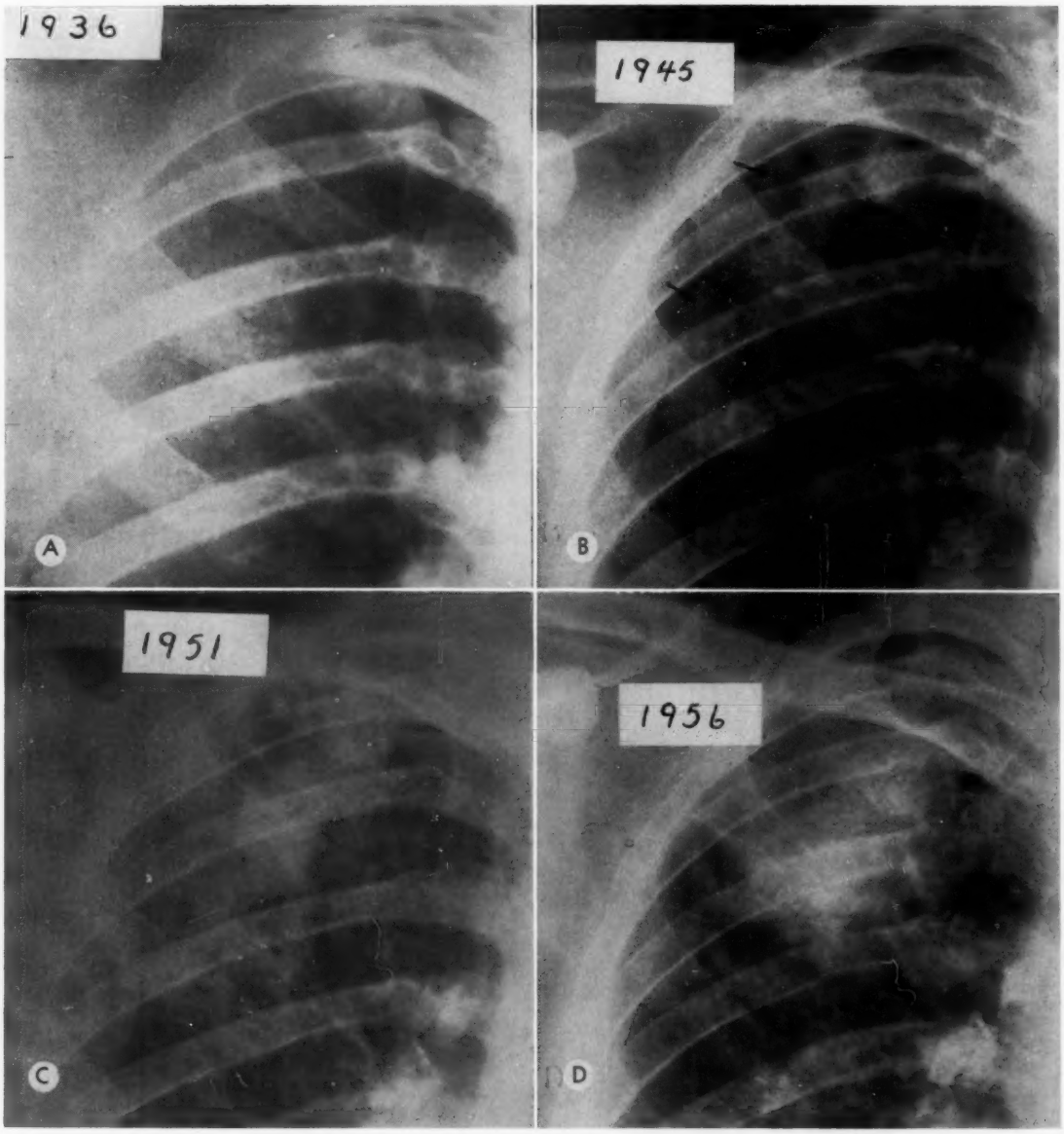


Fig. 18. Silicosis with infection, possibly tuberculosilicosis. (*A*) Appearance of right upper lobe in a silica worker, thirty-eight years of age in 1936. His chest was thought to be healthy. (*B*) This examination was made in 1945. A conglomerate shadow has appeared since early modulation developed in 1937. (*C*) Roentgenogram made in 1951. Note the progressive increase in the size of the lesions. (*D*) Roentgenogram twenty years after the individual first developed this lesion. Note the calcification in the hilus. We have never found any clinical or laboratory evidence of tuberculosis. He has not been skin tested with histoplasmin. This series illustrates the development of a massive lesion in a single individual who has had exposure to quartz dust with very little if any other dusts in the atmosphere.

tively different as is their pathology.

In the first group are those in which there is a fresh infection, either new or arising from the reactivation of a latent focus, superimposed upon a progressive and still active silicosis. The general tendency is toward uncontrollable extension to death before there is opportunity for the chronic

changes of tuberculosilicosis to occur. Rarely, several years may elapse before toxic symptoms supervene.

In the second group are the unusual but existent cases in which silicosis and tuberculosis occur together but act independently. The infection, observed by means of serial roentgenograms, is seen to behave typically as it does in nonsilicotic subjects.[70] The tuberculosis develops upon a background of an already stabilized silicosis in which the quartz particles are presumably completely isolated within their fibrous nodules and thus exert no effect on the superimposed infection. We have then simply the coexistence of silicosis and tuberculosis within the same patient, but without modification or acceleration of either disease process by the other.

Recently, we have observed another type. The patient was a relatively young man with widespread disease, which developed very rapidly. The distribution of the lesion was similar to that seen in miliary tuberculosis. The patient had tuberculosis of the larynx. Hoarseness was the chief complaint. The patient did not have any fever nor any signs of toxicity, but he had a positive biopsy and his sputum was positive for tubercle bacilli.

Chadwick and Pope[8] in discussing tuberculosis of "yesterday and today" call attention to the falling morbidity and mortality rates. The incidence of tuberculous infection and tuberculous disease is gradually decreasing. Cooper[14] states that this observation carries over into the silicosis group. Factors such as better nutrition, better housing, less crowding, and isolation of open cases of tuberculosis are responsible. Cooper[14] does not know of any good evidence to show that there is decreased virulence of the tubercle bacillus. He states that some phthisiologists have emphasized that the race as a whole shows improvement in resistance. Vorwald *et al.*,[82] in their studies of the effect of BCG* vaccination in guinea pigs with silicosis, demonstrate conclusively that the BCG strain used in their study can produce progressive and fatal pulmonary tuberculosis in guinea pigs whose tissue resistance has been modified by the inhalation of quartz dust. I know of no study of this type among silica workers.

* Bacillus Calmette-Guerin.

RAPIDLY DEVELOPING SILICOSIS

This type of silicosis[23] is not seen very often. When it occurs, there are two causative factors that seem responsible—gross overexposure to finely divided silica and a complicating infection, especially tuberculosis. On two occasions, I have had the opportunity to study serial roentgenograms in rapidly developing silicosis; one group was exposed in drilling a tunnel and the other while bagging silica sand. I have not seen a single case of this type within the past ten years.

The early roentgen appearance is that of a diffuse haze in the lower lobes, especially on the right side. The roentgenogram is not too informative, but the fluoroscopic study reveals some limitation of costal and diaphragmatic expansion.

In the more advanced lesions, there is evidence of lobar and/or lobular consolidation. In the chronic cases, the lower lobes show chronic scar contraction and the lung ventilation occurs largely in the upper lobes.

PULMONARY PHYSIOLOGY STUDIES IN SILICOSIS

Wright,[85] in summarizing his observations on pulmonary physiology in silicosis, states that the earliest form of silicosis, generally classified as simple discrete nodular silicosis, is attended either by no recognizable physiologic alterations of function of the cardiorespiratory apparatus, or, at most, by very slight ones. It is not surprising to me that such a statement is made, even though it may be modified when more information is available. The lungs have tremendous powers for compensation in health and disease.

When conglomerate silicosis develops, one usually finds alteration of pulmonary

function.[85] The maximal breathing capacity is usually reduced, the residual volume is increased, and the oxygen ventilation equivalent may be above normal.[85] The findings are like those found in diffuse obstructive emphysema. This type of abnormality can be demonstrated by fluoroscopy and roentgenography, but Wright[85] considers that for practical purposes it should be said that in all stages of silicosis, the severity of impairment of respiratory function bears only the grossest correlation with the extent of the disease found in the roentgenogram. This is not discouraging—there is an opportunity for the physiologist and the radiologist to pursue further investigative work.

ASBESTOSIS

The roentgen findings in asbestosis are seen first in the lower half of the thorax. This form of pneumoconiosis is notoriously difficult to diagnose during its so-called early stages. In fact, the patient may have symptoms of what in this country is often diagnosed a slowly-resolving virus pneumonia evidenced by weakness, chronic cough, but no fever. The roentgen findings in such patients after the disappearance of the pneumonia are essentially within normal limits so far as the fluoroscopic and roentgenographic observations are concerned. Yet, in such individuals, the microscopic studies of lung biopsies show thickening of the walls of the alveolar sacs and the respiratory bronchioles, and excellent examples of well-formed asbestos bodies (Fig. 19). My experience, therefore, is not in accord with that of Cartier[7] who says that "no cases of asbestosis of clinical importance have been diagnosed by the pathologist without having been detected anteriorly by the roentgenologist." I must hasten to say that I have not had lung biopsies on mine workers, but have had a few microscopic studies of lung biopsies on mill and textile laborers and on an individual whose work was concerned with covering pipes with asbestos for insulation purposes.

In answer to a query[73] as to the presence of asbestosis in a 1952 roentgenogram of a fifty year old man who had worked from 1940–1945 covering pipes with asbestos insulation, it was suggested that the exposure would have had to be much more severe in order to produce asbestosis. After seeing and examining a well-advanced and proved case of asbestosis in a man whose occupation was covering pipes with asbestos material, I find it difficult to express an opinion on just how much and what kind of exposure is necessary to produce asbestosis. For years, workers in covering pipes have not been thought of as working in an unusually dangerous dust hazard. The patient, referred to above, was a self-employed individual, intelligent, and although he knew asbestos dust could produce lung damage, he felt that he was not being exposed to enough asbestos dust to hurt himself. The dust hazard in this instance was produced by sawing and fitting the coverings (Fig. 20).

A careful fluoroscopic and roentgenographic study should be made of all workers going into the asbestos industry. A number of these workers are women, so one has to be concerned with the confusing shadows of the breast in women and heavy pectoral folds in men. Stereoscopic roentgenograms are helpful. Roentgenograms exposed in full inspiration and in full expiration in the posteroanterior and right lateral views, and posteroanterior views in each decubitus position will greatly assist as a baseline for future comparison in order to detect the earliest roentgen evidence of deviation from the normal. Oblique views (15 to 25 degrees) are most helpful in demonstrating pleural changes.

For the most part, the roentgen signs are not recognizable until the fibrosis has extended to and involved the visceral pleura. Early visceral pleural changes are not seen except at the interlobar fissures and then only when one makes special exposures in the cephalic or caudal views centered along the interlobar fissures (Fig. 21).

I have found the expiratory posteroanterior roentgenogram helpful in studying

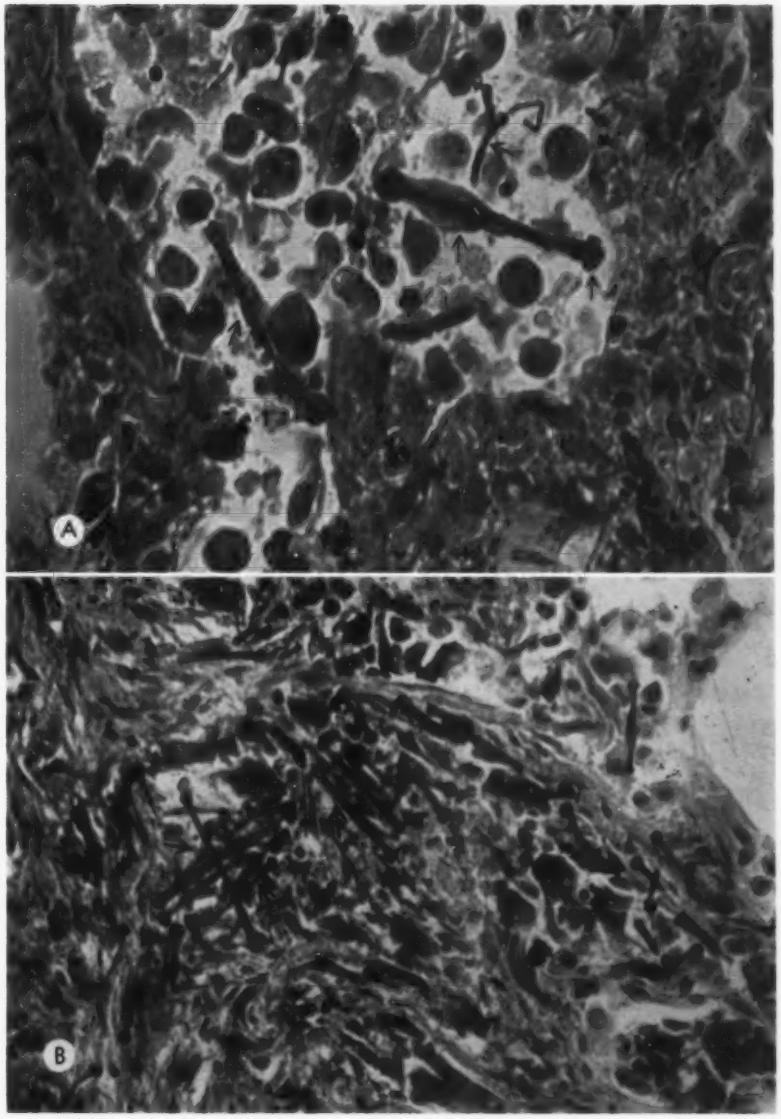


Fig. 19. (*A*) Several asbestos bodies in the lung. Note the various shapes (arrows) and the irregular deposit around the asbestos fiber. Some asbestos bodies have terminal globular swellings and transverse markings. (*B*) There are two large collections of asbestos bodies. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

asbestosis; in fact, sometimes it seems to provide more reassuring evidence of disease than is obtained on the inspiratory roentgenogram.

The earliest roentgen evidence of asbestosis is a presumptive limitation of diaphragmatic and costal expansion and roentgenographic evidence of restricted movement of the vascular shadows (elongation and separation of the truncal shadows). Such findings must be presumptive unless pre-employment studies are available for comparison. Retrospective studies are invaluable in asbestosis. Oftentimes the roentgen evidence of asbestosis is seen best in the left lower lobe. Just why this occurs is not known.

Gradual disappearance of the lateral costophrenic sulci is a helpful diagnostic sign in asbestosis and is interpreted as evidence

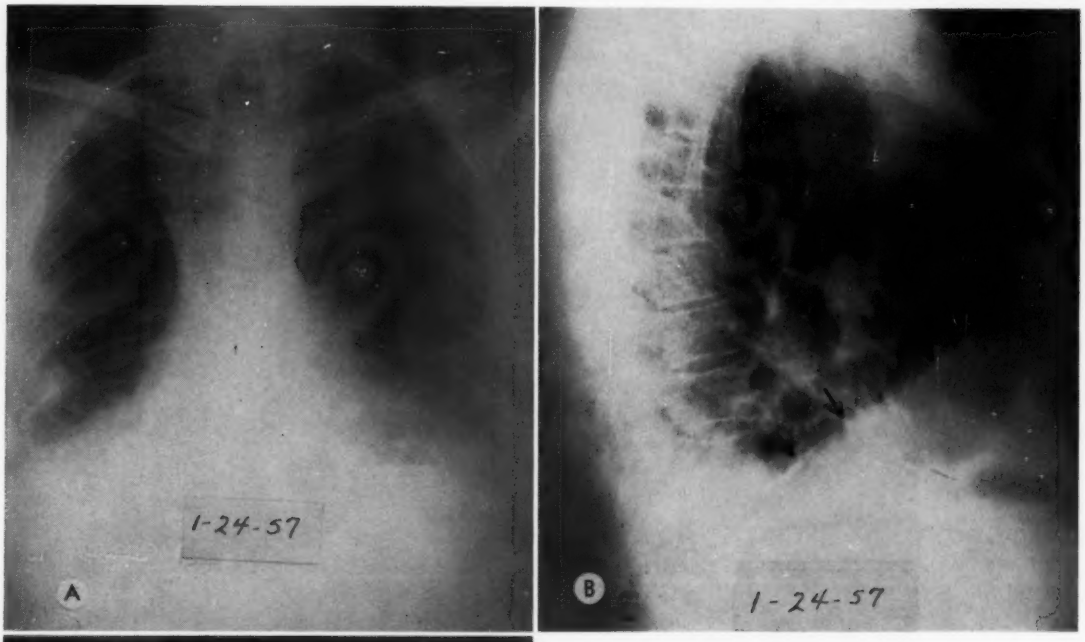
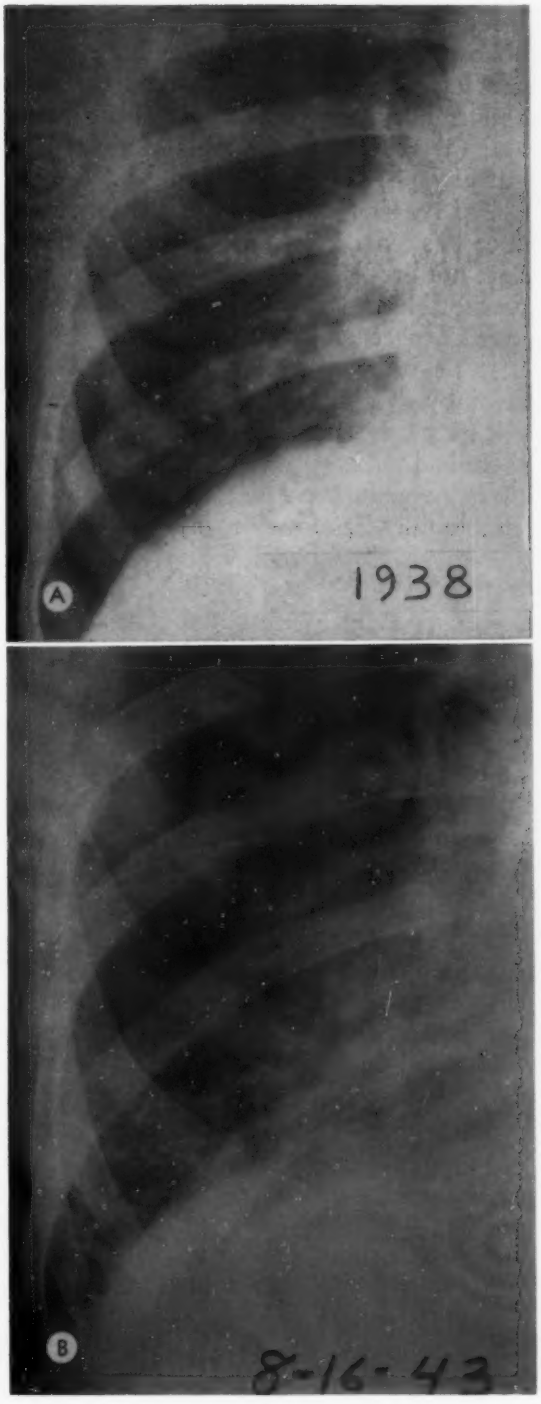


FIG. 20. (*A*) Advanced asbestosis in a man fifty years of age. This individual was self-employed and the exposure was caused by covering pipes with asbestos. (*B*) Lateral roentgenogram showing the tremendous thickening of the pleura. (*C*) Note the thickening of the pleura in the heavily exposed roentgenogram.

FIG. 21. (*A*) This worker is forty-one years of age. The roentgen findings are essentially negative. He has worked for ten years as a weaver. (*B*) Well advanced asbestosis in the same individual at forty-six years of age. He has now been a weaver for fifteen years. Note the marked changes in the right lower lobe and the difference in the roentgenographic appearance as compared to that observed five years previously. The changes in this individual are more prominent on the right side. The right dome of the diaphragm is elevated.

of pulmonary and pleural involvement. The sharp outline of the cardiac silhouette gradually becomes ill-defined due either to the blurring shadows of superimposed structures or thickening of the visceral pleura (Fig. 22). My explanation for the exaggerated pleural thickening in the lower lung areas is that the pleura seems to show greatest thickening in those areas where there is greatest mobility of the structures.



Possible trauma exerted by the fibers on the surrounding structures during respiratory movements is the real factor, not only in causing pleural thickening, but also in producing prominence of the left lower lobe markings; the cardiac pulsation may act as an additional factor.

In the advanced stages, the diaphragmatic excursion is markedly disturbed; the lower lung fields have an appearance of hypoventilation plus a leather-like or ground glass density. All structures, including the borders of the cardiac silhouette, are ill-defined (Fig. 23). The pleural shadows are greatly thickened, the thickening being less in the upper portions of the chest. The upper lung fields are ventilated, possibly hyperventilated, at times giving one the impression of emphysema. Asbestosis may produce shadows of a nodular pattern or irregular, stringy shadows not only in the lower lobes but in the middle or upper lung fields. The vertical diameter of the chest (first rib posteriorly to level of domes of diaphragm in inspiration) is reduced. Frequently, the elevation of the left dome is more pronounced.

Pulmonary Physiology in Asbestosis. Wright,[85] in his studies of asbestosis, showed that there is little and oftentimes no impairment of ability to ventilate the lungs in the typical asbestotic person, as measured by maximum breathing capacity. There is a slight to moderate reduction in total lung volume.[85] It should be possible to show this by fluoroscopy and with roentgenograms by demonstrating the restriction of thoracic and diaphragmatic movement. When lung fibrosis is extreme in extent, the maximum breathing capacity is limited, but fluoroscopy shows little or no impairment to emptying of the lung.[77,85] Gas studies of O_2 and CO_2 show an impediment to the passage of oxygen across the alveolar membrane, and during exercise the person with asbestosis overbreathes.[85] In spite of these and other complementary observations, Wright[85] indicates that it should be emphasized that his data show that an individual retains his average capacity for work in the presence of incomplete oxygenation of the blood. Wright,[85] in his discussion on asbestosis in connection with correlation of the roentgenologic and physiologic pulmonary studies, states—"that

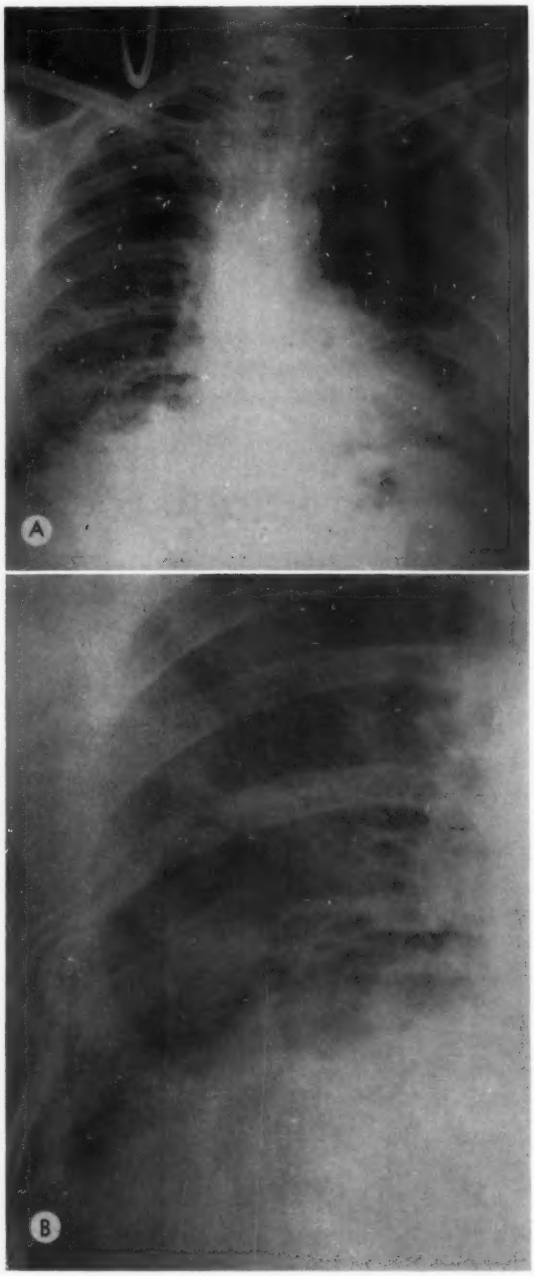


Fig. 22. (*A*) Asbestosis in a fifty-four year old man who worked as a laborer for four years operating a shaker which removed dust from the asbestos fiber. There was a mechanical ventilator in a large room with three windows and a door. This individual was in normal health until August, 1945 when he developed a dry hacking cough and dyspnea on exertion. There was a dull substernal pain. One can see evidence of fairly advanced changes in both lower lobes. There is definite thickening of the pleura on both sides with some obliteration of the costophrenic sulci. (*B*) The close-up study shows the marked changes in the right lower lobe. Movement of the domes of the diaphragm was fairly good in spite of the extensive lung changes. It was difficult to demonstrate good cardiac pulsations because of the overlying shadows in the lung field. The roentgen kymographic studies showed fairly good amplification of the pulsation of the heart.

three situations can exist: (1) physiologic abnormality without any definite roentgenologic abnormality, (2) roentgenologic abnormality plus physiologic abnormality, and (3) roentgenologic abnormality without any physiologic abnormality."

Tuberculosis and Asbestosis. My experience has led me to believe that asbestosis does not predispose or render a person more susceptible to tuberculosis. When tuberculosis does occur in an individual with asbestosis, its roentgen manifestations are essentially those of one who has had no complicating disease (Fig. 24). I am aware that there is difference of opinion about susceptibility in asbestosis and suspect that other factors may be of major importance.

COAL WORKERS' PNEUMOCONIOSIS

The pathologic studies of Hart *et al.*,[36] Gough,[30] Heppleston,[38] Gilson,[26] and the roentgenologic and epidemiologic studies of Fletcher and his scientific associates[21,22] have been responsible for a new concept in pneumoconiosis. If Collis[12] could examine the pathologic and epidemiologic data that have been collected by Gough and his colleagues, I feel sure that he would modify the statement contained in his scholarly Milroy Lecture which reads as follows: "some dusts, such as coal, it is true, not only appear to have no power of producing pneumoconiosis, but even may possess some inhibitory influences on phthisis; . . ."

Re-orientation concerning the pneumoconiosis problem among the anthracite and bituminous coal workers has been difficult for many of us, who, for years, have been disciples of those who believed preponderantly that disability was the result of the fibrogenetic effects of finely divided silica. Kammer[42] feels that we in America should re-assess the whole problem of the miner. I

would be inclined to extend the re-assessment to workers in other industries—two examples being the talc workers in the State of New York, and the gold miners in Western Australia.[1]

The work of Gough and his colleagues is most important in another respect. There are many well-trained young men in industry, public health, and medicine, who have new techniques which extend beyond the limits of the microscope; beyond the previous concepts of acceptable dust counts for a healthy atmosphere; and, beyond old ideas on environment. Lastly, but not least, they cannot be as completely dedicated to silica as I found operative in my own thinking. The future for better working and living conditions should be bright.

It should be emphasized that many of the coal miners in this country have had exposures to silica from hard rock mining and the pathologic process in their lungs may not correspond to that under discussion. Their disease is one of a modified silicosis, *i.e.*, anthracosilicosis. That form of pneumoconiosis is not being considered here.

Coal workers' pneumoconiosis is not a new condition. It occurs largely among those who work at the coal-face, but it occurs elsewhere also, even among those loading the coal into the ships.[26]

In the pathologic studies of Hart *et al.*,[36] Gough,[30] and Heppleston,[38] it was noted that the condition is pathologically distinct from silicosis. The lungs contain large quantities of coal dust, which is aggregated into foci about the respiratory bronchioles. The fibrosis is sparse, the coal dust being held in a fine mesh of reticulin fibrils, stellate in appearance. The second finding was that the pathologic process was the same in every coal field, suggesting that there is nothing biologically peculiar about anthracite. The third finding was that the disease has two forms. First, simple pneumoconiosis, which is characterized by small foci of coal distributed throughout the lungs, these foci being surrounded by small areas of emphysema, so-called focal emphysema. Secondly, infective pneumoconi-

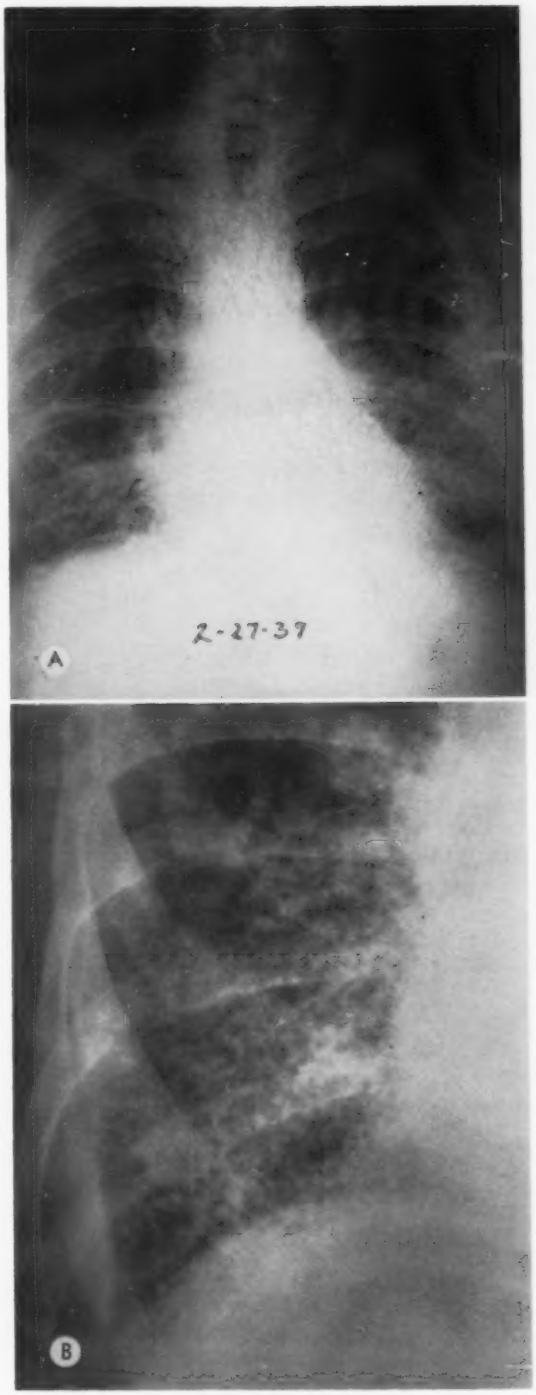


Fig. 23. (*A*) Well advanced asbestosis in an individual fifty-three years of age who had worked for over thirty years with asbestos. He had clubbing of the fingers, chronic cough, dyspnea, anorexia and fatigue. Note the pattern of the disease throughout both lungs, especially in the left lower lobe. (*B*) The close-up study illustrates very well the changes in the lung and shows the thickening of the pleura along the right lateral chest wall.

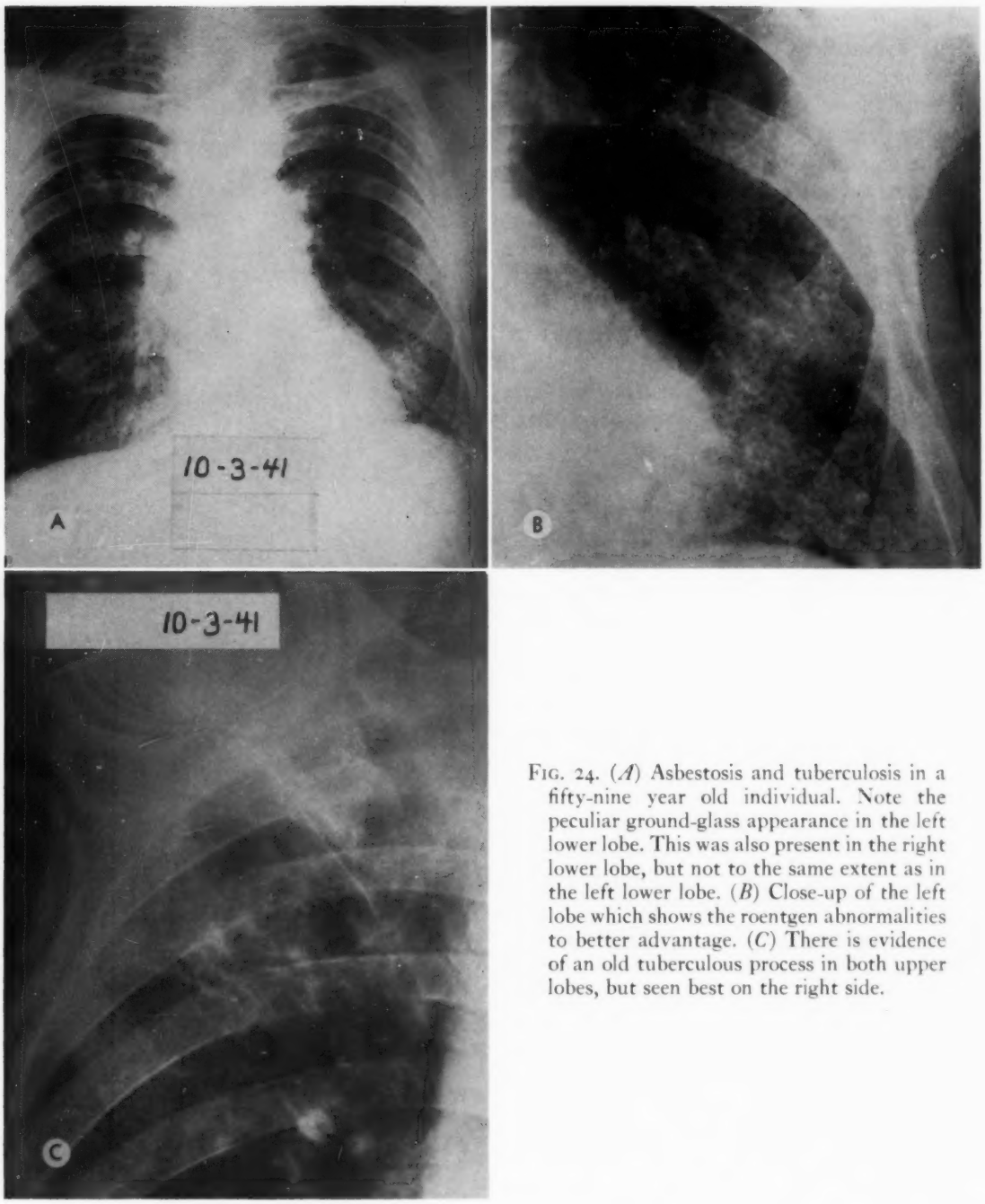


Fig. 24. (*A*) Asbestosis and tuberculosis in a fifty-nine year old individual. Note the peculiar ground-glass appearance in the left lower lobe. This was also present in the right lower lobe, but not to the same extent as in the left lower lobe. (*B*) Close-up of the left lobe which shows the roentgen abnormalities to better advantage. (*C*) There is evidence of an old tuberculous process in both upper lobes, but seen best on the right side.

osis, which starts as a collagen fibrosis within a few coal foci and subsequently enlarges and coalesces to form a dense mass of fibrous tissue occupying much of a lobe or even a whole lung. This second form is thought to be due to tuberculosis superimposed upon a lung heavily laden with coal dust.

The roentgenologic manifestations are classified as follows:[20] *Simple pneumoconiosis* refers to those cases having minute shadows throughout both lungs. Simple

pneumoconiosis is subdivided into three categories of increasing profusion of the discrete shadows. *Complicated pneumoconiosis* refers to those cases having shadows of larger size. Complicated pneumoconiosis is divided into four categories: (*A*) Shadows occupying at least one anterior rib space, generally more or less circular, 1 cm. in diameter and of uneven density; (*B*) dense shadows more extensive, better defined and more homogeneous than category *A*, and occupying less than three anterior rib spaces; (*C*) similar shadows extending more than three anterior rib spaces; and (*D*) shadows extending more than three anterior rib spaces plus gross distortion of the pulmonary architecture.

There are two disease hypotheses. The studies suggest that man's environment shows differences in the natural history of simple and complicated pneumoconiosis. *Simple pneumoconiosis* only appeared and progressed as such in those who were exposed to coal dust. *Complicated pneumoconiosis* appeared and progressed in those who left the mine; it did not appear to any extent in those who had less simple pneumoconiosis than would be classified in the second category.

In surveys on coal miners, McCallum and Browne[54] found in the roentgenographed group that the roentgen evidence of pneumoconiosis increases in prevalence with rising age and with length of time spent at facework. Between twenty-six and thirty-five years of age, nearly 8 per cent had simple pneumoconiosis and the percentage of men affected rose sharply between thirty-six and forty-five years of age. These two age periods correspond roughly to from one to ten years and eleven to twenty years at the face. They observed that the length of time at the facework is more important in producing roentgen signs of pneumoconiosis than is the period of time working in the pit. These authors believe that the men with pneumonconiosis should be allowed to continue work at the face only under medical supervision.

Clinical studies[43,47,51,52,76] on coal workers in this country continue and in at least two communities,[47,52] the influence of our English colleagues has become manifest. The use of the "standard set" of diagnostic roentgenograms is being employed.

In studies concerned with the relationship between *tuberculosis* and *pneumoconiosis* in coal miners, Cochrane[9] suggests "that a little coal dust retained in the lung has a definite therapeutic effect, whereas larger accumulations increase the attack and mortality rates from tuberculosis." It is felt that with "standard film guides,"[54] the roentgenologic diagnosis of tuberculosis can be made with reasonable consistency and that shadows characteristic of tuberculosis can be distinguished in roentgenograms from those of simple and complicated pneumoconiosis.

The attack rate of the re-infection type of tuberculosis is found to be related to the severity of the associated simple pneumoconiosis[50] and is higher than in the general population. It seems to be reduced in cases of complicated pneumoconiosis.

For comparative purposes, it is of interest to note that Wall,[83] in a study of 100 autopsied cases of anthracosilicosis, found pulmonary tuberculosis in 3 cases, as judged by histopathologic criteria and examination for acid-fast bacilli on stained smears. The majority of the patients died of heart disease, with cor pulmonale accounting for one-half of the cardiac deaths.

Pulmonary Function and Roentgenologic Correlation. Gilson and Hugh-Jones,[27] in 1949, undertook an investigation with the object of determining the precise cause of breathlessness in a group of coal workers and relating its severity to the roentgen changes in the lungs. Their results showed that the main disability from pneumoconiosis, excessive breathlessness on exertion, cannot be distinguished by functional tests from breathlessness caused by chronic non-industrial pulmonary disease. This observation renders it difficult to allocate in an individual the proportion of breathlessness caused by industrial exposure.

In England, the National Insurance (In-

dustrial Injuries) Act (1946) attempts to make such an allocation. Under its regulation there are two separate issues in relation to the disability benefit for pneumoconiosis: first, its diagnosis, "has this man got pneumoconiosis within the meaning of the Act?"; secondly, the disability assessment, "to what extent is the man disabled and what proportion of his disability is due to pneumoconiosis?"

Under the Industrial Injuries Act in England, the definition of pneumoconiosis is based on pathology and during life the diagnosis is based on the occupational history and so-called "characteristic" roentgen appearances. Unfortunately, the correlation between roentgenologic and pathologic findings is not perfect. But, the studies of Gilson and Hugh-Jones[27] emphasize two aspects concerning the roentgen diagnosis. First, miners with no roentgen evidence of pneumoconiosis are, on the average, as breathless for their age as men with simple pneumoconiosis and more breathless than non-miners, particularly the elderly. This suggested that such miners might logically receive benefit for their disability. Secondly, functional emphysema, which increases with the ascending roentgenographic categories, is part of the same pathologic process described for pneumoconiosis. Under those circumstances, it is regarded as illogical to give extra disability benefit for "emphysema" to disabled men who have the roentgenographic changes of pneumoconiosis.

In *simple pneumoconiosis* there was no average increase in breathlessness on exertion with increase in roentgenographic abnormalities. However, there was an increase in breathlessness normally associated with age, old men being proportionately more breathless than young men whatever the degree of change.

In *complicated pneumoconiosis*, the breathlessness was nearly always severe and increased with the roentgenographic changes, and was augmented even more by advancing age than in simple pneumoconiosis.

In assessing the extent of disability, it is necessary to consider all disabilities as they relate to "loss of health, strength and power to enjoy life." The extent of breathlessness was the main disability to receive Gilson and Hugh-Jones' consideration, and the one which we as radiologists are most concerned about. Their studies indicated that a combination of the standard exercise test and some determination of the maximum ventilatory capacity gave the best objective measure, but that it was probably unsuitable for the Pneumoconiosis Panels. Gilson and Hugh-Jones suggest that possibly the simple procedure of measuring diaphragmatic movement and level is well enough related to maximum ventilation to allow a reproducible division of subjects into 4 or 5 grades pertaining to breathlessness. Their most discriminating test permitted the division of range of breathlessness between normal and extreme disability into nine steps. These authors indicated that there are clear differences between the average degree of breathlessness for each roentgenographic stage and the normals at the same age. They stated: "This 'average' increase of breathlessness at each x-ray stage might justifiably be regarded as that due to pneumoconiosis, and a case could be made out for allotting benefit for 'disability due to pneumoconiosis' simply upon the x-ray stage at a given age, and this is the most practicable and logical conclusion to draw from our results."[27] The work of Gilson and Hugh-Jones is a magnificent contribution and goes a long way to show the possibilities in this field.

Mobility of Pneumoconiotic Deposits. In 1918, Haythorn[37] noted that in an anthracotic lung there was always a large number of free pigment-bearing cells during the early stages of pneumonia. This observation, as well as his continuing interest in the subject, was somewhat esoteric to me —a young radiologist, because it was thought to be impossible for one to demonstrate the roentgen manifestations of the results of migration of alveolar phagocytes loaded with dust particles. The clinician,

on the other hand, observed that the coal miner continues to have a black sputum for a long time after he gives up work in the mine. Such observations, as well as clinical improvement of some workers after leaving dusty occupations are to be regarded as significant even if one does not become interested in basic physiologic processes.

Fortunately, there are investigators[34,35] who do appreciate the need for basic work with the excursionary activity of alveoli, the mechanism by which dust is removed from the interior of the lung, and the implications that such knowledge may contribute to solving some of the problems of pneumoconiosis.

I now believe that the radiologist may be able to make a minor observation concerning the mobility of pneumoconiotic deposits, possibly in welders and workers in other opaque dusty occupations such as barium and tin oxide workers. This opinion is based on the observation of a considerable disappearance of the pattern of hemosiderosis in mitral heart disease following commissurotomy. Follow-up chest roentgenographic studies on individuals like welders, after they are removed from their dust exposure, may provide interesting data on what can be expected from the roentgenologic standpoint.

PNEUMOCONIOSIS AND RHEUMATOID ARTHRITIS (CAPLAN'S SYNDROME)

In 1953, Caplan[6] called attention to an association of unusual massive lesions in the lungs of coal miners with rheumatoid arthritis. He found an increased frequency of massive lesions in these arthritic persons, and, in some, the massive lesions were peculiar in that they were multiple, round, well defined and distributed rather evenly throughout the lungs. In others, the roentgenologic appearance resembled the usual massive lesions of infective pneumoconiosis. The lesions were not associated with constitutional symptoms nor unusual respiratory distress. Caplan does not suggest that all coal miners with pneumoconiosis and rheumatoid arthritis show a characteristic roentgenologic appearance.

Subsequent to Caplan's observations, others have made additional studies.[33,56,72] Gough, Rivers and Seal[33] have reported on the pathologic findings of the lungs of 14 Welsh coal miners with rheumatoid arthritis and biopsy lung specimens from 2 additional cases. They concluded: "Nodules having a distinctive gross appearance correspond with the radiographic round opacities described by Caplan (Fig. 25). These 'rheumatoid' pneumoconiotic nodules contain necrotic collagen and dust. Tuberculosis was present in several cases. A nonspecific inflammation was also found. This is believed to be the 'rheumatoid' component."

Miall[56] has carried out an epidemiologic survey of rheumatic arthritis in a complete community of 9,430 males over fifteen years of age in South Wales and among other things observed that there was a significantly high prevalence of massive fibrosis and tuberculosis among miners with arthritis. Price and Skelton[72] have described widespread nodular lesions in the lungs of a woman, aged fifty-eight, with rheumatoid arthritis. At necropsy, scattered focal lesions were found in the lung, in which there was an active subacute inflammation of the arteries and bronchioles.

The foregoing comments are only a few of the reports relating to some aspect of the possible implication of Caplan's observation (Caplan's syndrome).

We have not made any particular study on the subject of rheumatoid arthritis and pneumoconiosis, although we are interested in any factor which may have a modifying influence on the lesions of pneumoconiosis. Therefore, in an informal manner, we have been collecting roentgen studies on individual workers and patients who have rheumatoid arthritis and pneumoconiosis. I am not prepared at this time to do more than say that I have seen roentgenograms of individuals exposed to relatively small amounts of coal dust and silica which demonstrated lesions similar to those described by Caplan and others. In some of the lesions there was evidence of calcium

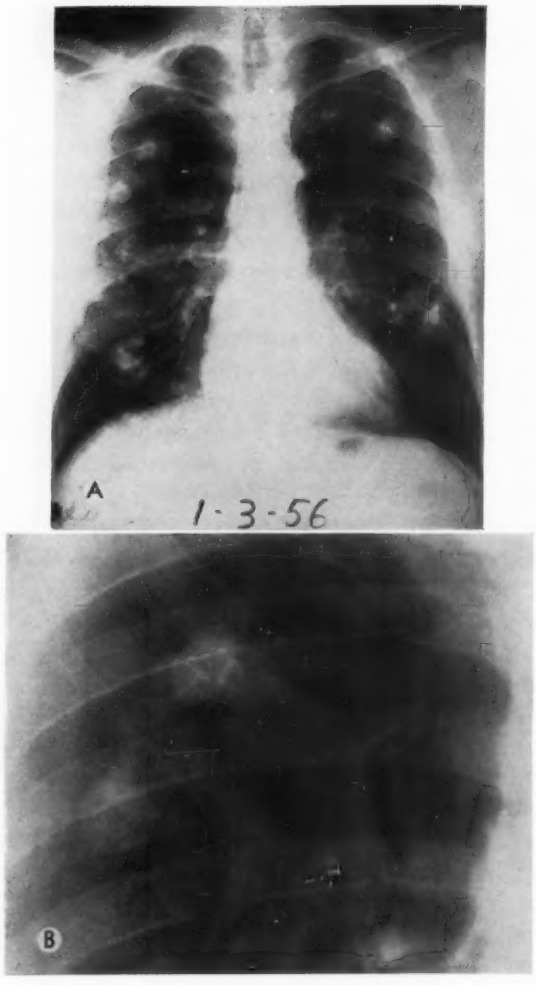


Fig. 25. (*A*) Caplan's syndrome (coal workers' pneumoconiosis with rheumatoid arthritis) in a fifty-six year old individual. This person has been exposed to coal dust for seven years and he has a well advanced rheumatoid arthritis. The diagnosis was made by roentgen examination and has not been confirmed by lung biopsy. (*B*) Close-up of lesions in the right upper lobe. The lesions look very much like malignant metastatic nodes, but this individual has been under observation for some six or seven years and we believe that malignancy can be excluded on the basis that they have not changed in size.

deposition. Whether the calcium was resultant from tuberculosis, histoplasmosis or some other necrotizing process is uncertain (Fig. 25).

SILICOSIS AND PULMONARY CARCINOMA

The radiologist in a general hospital is unable to give an opinion on the incidence of carcinoma of the lung. His patients are usually referred for definitive therapy and occasionally for diagnosis. The radiologist is, however, confronted with a diagnostic problem when he makes an examination of the chest of an individual who has silicosis and who has symptoms that may be interpreted clinically as suspicious of carcinoma.

I have seen silicotic persons who have had persistent hemoptysis without definitive lung lesions which one would interpret as either tuberculosis or carcinoma. Subsequent bronchograms have demonstrated stenosed bronchi and at operation the stenosis was the result of fibrosis and scar contraction and not due to carcinoma. On other occasions, I have seen patients with hemoptysis, unilateral massive lesions, and suspicious Papanicolaou smears. No carcinoma was found at operation.

When the silicotic patient has multiple conglomerate lesions with a pattern of nodulation, it is impossible or at least improbable that one can make a roentgen diagnosis early enough for a curative surgical procedure. If there is any doubt as to the identity of the lesion, one should utilize all diagnostic means, including bronchography, selective angiography, and Papanicolaou smears. If one is still uncertain, we recommend surgical exploration.

We have noted that a number of silicotic patients often are referred directly to the thoracic surgeon with a presumptive diagnosis of carcinoma of the lung. These patients have frequently been either hard or soft coal miners. In every instance with 2 exceptions, the roentgen diagnosis has been silicosis with infection. One of the exceptions, diagnosed as carcinoma, showed a narrow and funnel-shaped bronchus surrounded by a mass. The lesion was a silicotic mass. The other exception was a lobar consolidation, typical of atelectasis from a bronchial obstruction. That was a carcinoma.

The physician, in any discipline, who is unfamiliar with some of the vagaries of silicosis and other forms of pneumoconiosis

will conclude that a unilateral conglomerate or massive lesion in the absence of a pattern of nodulation should be regarded as carcinoma until proved otherwise. That is especially true if there is a high index of suspicion for carcinoma of the lung. I have found laminagraphy most helpful when in doubt about the nature of a massive lesion in pneumoconiosis. Distortion of bronchi, calcium deposition, small emphysematous areas around the lesions, and possibly other small silicotic lesions assist in excluding carcinoma as the first choice for diagnosis.

There are two types of carcinoma seen in patients with silicosis that frequently are misdiagnosed. One group includes those squamous carcinomas which become necrotic in the center, break down and subsequently the patient coughs out most of the necrotic material leaving a fairly thick-walled, large, oval cavity, which gradually increases in size. One rarely, if ever, finds tubercle bacilli in the sputum. The lesion may or may not extend to and destroy the adjacent ribs (Fig. 26). Oftentimes, the patient does not complain of much pain, even though, the ribs are eroded. Because of the large cavity and rib erosion and silicosis, there is an unbelievable urge to call such lesions tuberculosis. Tuberculous cavities are either thin walled or, as so often happens, thick walled. The cavity is irregular and is rarely larger than 5 to 10 cm. in diameter. The cavity noted in primary carcinoma of the lung is usually 5 or more cm. in diameter when first seen. There is practically no reactive lung lesion around the wall of the cavity.

The second group of carcinomas that are likely to be missed includes those that develop slowly amid shadows which may have been interpreted as due to silicosis with infection. Carcinoma is not suspected but tuberculosis is thought to be the presumptive modifying infection. When the carcinoma has grown large enough, the bronchus is obstructed and atelectasis takes place. The atelectasis produces a sudden increase in the roentgen manifestations. Such rapid progression of the shadow density in the roentgenogram, rarely, if ever, takes place in tuberculosilicosis.

Lung lesions which have migrated to the hili produce shadows that look not unlike lymphoblastomas or other mediastinal tumors. When dealing with pneumoconiosis, one should adhere strictly to a rule of not giving radiation or chemotherapy for a presumed diagnosis of malignancy. Biopsies for confirmation of the diagnosis are recommended; otherwise, tremendous damage to the tissue might occur.

Under pathologic considerations, comment has been made on the incidence of carcinoma of the lung among silicotic persons, but there are several other studies that should be mentioned. Schoch[75] in an investigation of material from the Swiss accident insurance organization found no etiologic relationship between silicosis and primary carcinoma of the lung. Wall,[83] in a postmortem study of a series of 100 cases of anthracosilicosis, found 4 cases of primary carcinoma of the lung. James[41] reports that primary lung carcinoma was found at necropsy in 3.3 per cent of 1,827 South Wales coal miners and in 5.4 per cent of 1,531 South Wales non-miners. In 12, the tumor was in the mass, and touching it, in 5. In 7, the tumor was remote. The tumor was similar in the histologic variants, in the age incidence, and in the distribution of metastases as that seen in the non-miners.

ASBESTOSIS AND PULMONARY CARCINOMA

I personally have not studied any cases of carcinoma of the lung in asbestosis. The roentgen studies of the cases that I have seen have not been serial studies, but have been roentgenograms of advanced lesions, such as atelectasis of a lobe, a large mass adjacent to the hilus or a unilateral lesion in an upper lobe. Lynch and Pratt-Thomas[49] indicate that the statistical evidence for a causal relation between lung carcinoma and asbestosis is strong. An opposite opinion is expressed by Jacob and Bohlig.[40] In Dresden, carcinoma among asbestos workers seems to be rare. Four cases of fatal carcinoma of the lung were found in 339

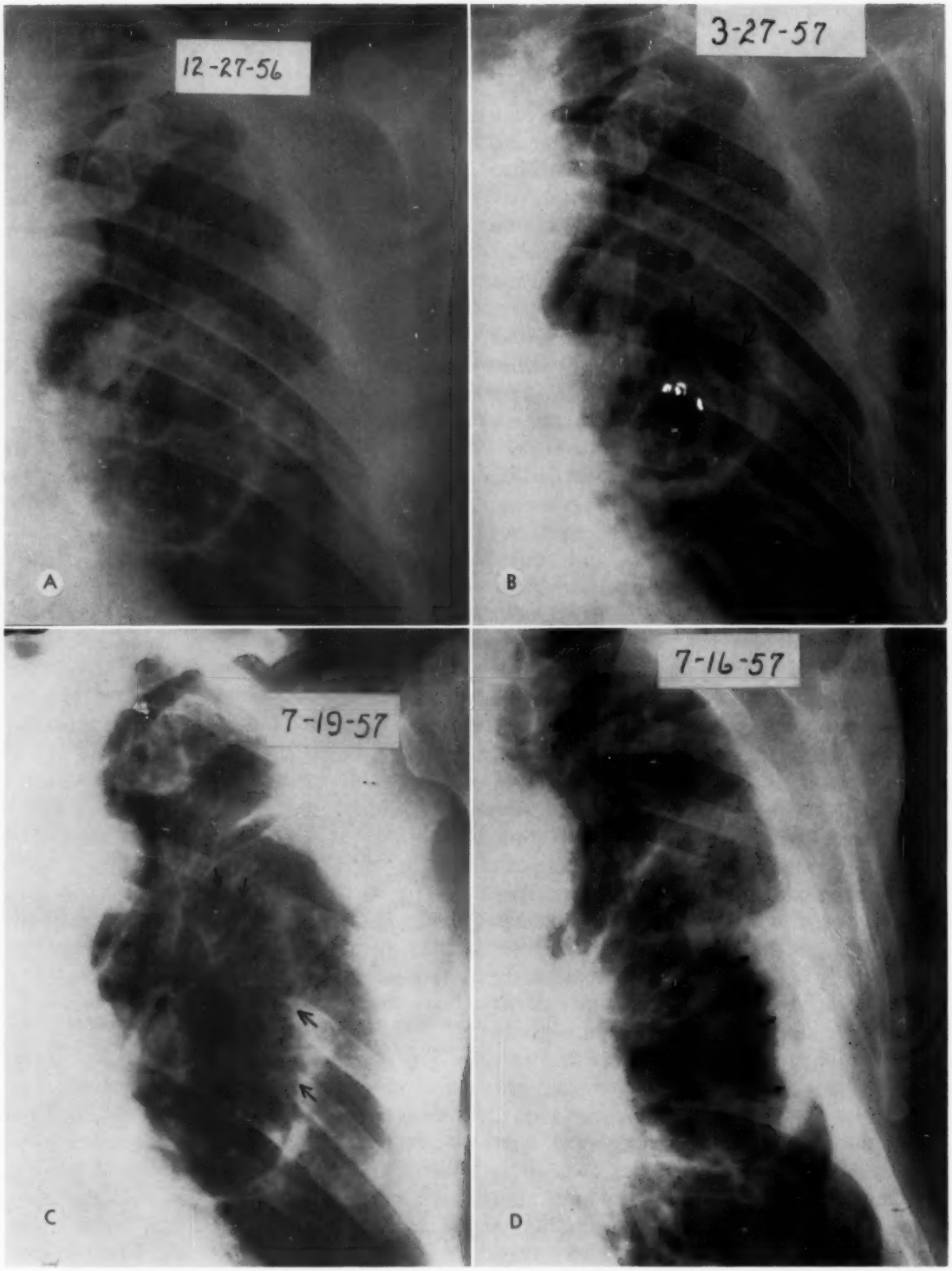


FIG. 26. (*A*) Carcinoma of the left lung in a seventy-one year old white man who had been a coal miner for forty years. He had been in his usual health except for increasing shortness of breath and chronic cough until November, 1956, when he developed pain in the left side of the chest and hemoptysis. A roentgen examination on December 27, 1956 showed a large cavity in the upper portion of his lower lobe. It was thought to be tuberculosis and anthracosilicosis. Note that the ribs are not involved. (*B*) Roentgenogram made in March, 1957. The appearance was essentially the same except that this time the sixth rib was eroded. (*C*) Roentgenogram made in July, 1957. The cavity was larger and portions of four ribs were destroyed. We thought the lesion was a carcinoma which had largely excavated itself. A biopsy confirmed the diagnosis. (*D*) A slightly oblique roentgenogram shows the wall of the lesion adjacent to the thoracic wall (arrows).

asbestos workers from 1930 to 1954. Doll,[15] in a study of 105 autopsies of asbestos workers, found 15 carcinomas of the lung associated with asbestosis. He concludes that lung carcinoma is a specific industrial hazard of certain asbestos workers and that the average risk among men employed twenty years or longer is 10 times that experienced by the general population.

PNEUMOCONIOSIS AND PULMONARY CARCINOMA

Carcinoma of the lung is a major cause of death today; therefore, the relationship of environmental factors to the etiology of lung cancer is receiving increasing consideration. Epidemiologic studies have been carried out in many industries, and are likely to increase, as guide-lines are evolved and a program of confidence is developed between governmental agencies, labor, and industry.

SUMMARY AND CONCLUSION

1. Pneumoconiosis continues to be a health hazard, national and international in scope.

2. There are many dedicated individuals in science, in medicine, in organized labor, in industry, in insurance, in the legal profession, and in a number of agencies in our local, state and national governments who would welcome a united effort to prevent pneumoconiosis and its complications.

3. Laws governing work conditions and workmen's compensation have been helpful; but, organized continuing education is likely to be a better approach in this country because, unlike England, we have 48 States, most of which have different codes and laws. In 1936, the United States Secretary of Labor, Frances Perkins, recognized the need for an organized educational effort to provide industry, labor, medicine, and government with an informed opinion concerning silicosis. After the several committees wrote their reports[57,58,59,60,61] for publication, the committees were discharged.

Conditions have changed since the publication of the Perkins committees' reports. Many of the industrial processes elaborating dust are new. Scientists now believe that submicroscopic dusts should receive major consideration. It may be necessary to re-evaluate the concepts concerning the amounts of dust that constitute safe levels. Methods of dust counting may need revision. Development of aerosols for control of dust hazards seems to be increasingly effective. There are many, many other phases of this problem which involve careful consideration by disciplines outside of radiology, but radiology has an important task to perform.

4. The radiologist plays a major role in the diagnosis of pneumoconiosis in the living individual. He assumes a role of increasing importance in the estimation of disability and in the results of rehabilitation.

It is said that the majority of controverted compensation cases involve questions of fact rather than questions of law.[39] Most of the fact cases involve a medical question.[39] Some of the questions are: What is the diagnosis? When does the condition become disabling?[84] When should the employee, subject to exposure, be removed from his employment?[84] When should such employee be deprived of his privilege of continuing at work in the occupation in which he may be engaged?[84] What methods are available to medical science to pronounce that the employee is totally disabled?[84] What sequelae result from a diagnosis of partial disability?[84] Should the employee at that time terminate his employment?[84] These are some of the imponderables with which the physician and the lawyer are faced in their consideration of pneumoconiosis in an individual seeking compensation. It is estimated that workmen's compensation administration is about 80 per cent medical.[39]

The satisfactory operation of the workmen's compensation system depends largely on competent and adequate medical treatment of injured employees, the prompt furnishing of accurate medical reports on injuries and the giving of medical

testimony at hearings on controverted claims. In compensation cases, the physician's responsibility is not confined to the patient but extends to the community, in the persons of the members of the industrial accident commission and to the involved industry.[44] The radiologist will be able to serve the patient and the community better if the profession can agree on guide-lines of diagnosis and extent of disability. This has been done in England and other countries. We should be able to develop working guide-lines for the United States. Guide-lines for diagnosis and disability ultimately would provide essential data for the scientist experimenting with new techniques for humans and the biometrician analyzing epidemiologic and environmental data of communities where there are dust hazards.

It would provide information for the State Pneumoconiosis Panels and industrial accident commissioners which should not be susceptible to as much disagreement among medical experts as obtains now.

Our present method of medical education does not provide for the implementation and the follow-up information that are necessary in controlling pneumoconiosis. Often times, medical practice does not keep pace with new and acceptable concepts, especially, when new data develop within a discipline outside of medicine.

5. Some of the other groups concerned with the problem of pneumoconiosis include: the general physician; the internist; the cardiologist; the pulmonary physiologist; the allergist; the phthisiologist; the thoracic surgeon; the bronchologist; the physical therapist (rehabilitation); the pathologist; the experimental pathologist; the public health officers; members of Bureau of Standards; United States Department of Labor; the industrial hygienists; the air pollution engineers (ventilation, aerosols, etc.); the industrial physicians; the psychiatrist; the representatives of labor, industry and insurance; the lawyers; the physicist; the biometrician; and the toxicologist.

6. It is suggested that the American Roentgen Ray Society initiate through its President and its Executive Council a proposal that the National Academy of Sciences request the Committee on Radiology of the Division of Medical Sciences of the National Research Council to formulate plans for the inauguration of a *National Committee on Pneumoconiosis Protection and/or Control.* A prototype of such a committee is the National Committee on Radiation Protection. The National Committee on Radiation Protection is so constituted that all interested groups are represented. It is a continuing committee which keeps informed on new developments and, with similar committees from other countries, agrees on acceptable methods and standards of good practice. Its reports are published in scientific journals and handbooks and are made available to everyone concerned. These reports are educational and informative, but are not regulatory.

7. Finally, if we as radiologists will accept the challenge presented by the problem of pneumoconiosis and do something constructive about it, we can feel assured that the ideals and conquering spirit of Caldwell and other great pioneers, indeed, will continue to live.

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POSTOPERATIVE ASPIRATION PNEUMONITIS

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ASPIRATION during anesthesia is not the serious complication that it was twenty years ago because of the current widespread use of effective antibiotics and improved anesthetic methods. However, in certain instances aspiration will lead to pneumonitis, pulmonary edema, shock, heart failure, and death despite modern methods of treatment. Vomiting and aspiration of gastric contents occurs frequently in obstetric patients admitted for prompt delivery on a full stomach, in traumatic fracture cases requiring anesthesia for immediate reduction, and in those who for one reason or another undergo immediate surgical procedures soon after admission. This is not to infer that the elective surgical patient will not regurgitate and aspirate during anesthesia. Berson and Adriani[1] report a 7 per cent incidence of aspiration in patients undergoing elective surgery with general anesthesia. Therefore, the diagnosis should be considered in all postanesthetic patients who develop positive pulmonary clinical and roentgenographic findings.

The purpose of this article is to present a brief review of the literature with case reports including salient roentgenographic findings.

REPORT OF CASES

The following cases were seen at the Hartford Hospital during a seventeen month period between April 12, 1956 and September 20, 1957. These are selected, not necessarily representing all such cases during this period.

CASE I. A seventy-four year old white female was admitted on September 16, 1956 with fracture of the left hip. The hip was pinned on the following day and her immediate postoperative course was uneventful. While convalescing, she developed signs of cholecystitis and on October 9, 1956 a cholecystectomy and common duct exploration were performed. Her postoperative course during the first twenty-four hours was uneventful, but shortly thereafter her temperature rose; she developed labored breathing with tracheal gurgles and rapid respirations and pulse. The patient was cyanotic. These changes were the result of an episode of regurgitation and aspiration. The trachea was suctioned on several occasions by the anesthetist and the patient was bronchoscoped on one occasion. However, despite all measures her course was retrogressive to death within twelve hours.

A portable roentgenogram of the chest obtained shortly before death showed patchy areas of increased density with associated atelectasis of the posterior basal segment of the right lower lobe. There was also elevation of the right hemidiaphragm (Fig. 1).

CASE II. A twenty-six year old white female gravida III, para II, entered the hospital on April 12, 1956 after an uneventful prenatal course. There was a normal labor of five and one-half hours' duration and successful de-

FIG. 1. Case I. A seventy-four year old patient after cholecystectomy with common duct exploration. Symptoms of cyanosis and labored breathing twenty-four hours following laparotomy. Bedside chest roentgenogram shows atelectasis of the posterior basal segment of the right lower lobe, at *a*. Note elevation of the right hemidiaphragm and absence of silhouette sign (Felson).

livery under general anesthesia. Emesis and mild cyanosis were recorded by the anesthetist during the procedure. Post partum she complained of a sore throat and had a severe cough. There was no temperature elevation. Roentgenograms of the chest revealed irregular areas of consolidation posteriorly in the right upper lobe. This patient received no specific therapy and repeat roentgenograms three days later disclosed nearly complete clearing of the above described changes (Fig. 2).

Case III. A twenty-eight year old white female prima gravida with known multiple sclerosis was admitted on April 19, 1957 in labor. At that time she showed a 1 plus albuminuria, peripheral edema and a history of 25 pounds weight-gain during pregnancy. Approximately six hours after admission she had a grand mal seizure controlled by pentothal. She was then taken to the operating room for a cesarean section. Upon arrival, she was "crowing due to laryngeal spasm and exhibited mild cyanosis." A nasotracheal tube was passed blindly and bright red blood was suctioned from the trachea. The trachea was lavaged and a few blood clots were obtained.

The cesarean section was performed without incident, except that she vomited some gastric contents. However, there was no obvious aspiration. For the first postoperative week she was in a precarious condition, requiring constant oxygen, fog, numerous tracheal aspirations, tracheotomy and a host of medications.

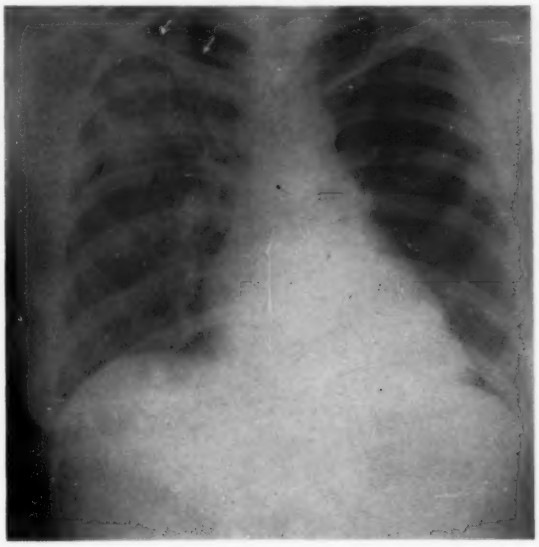

Fig. 2. Case II. A twenty-six year old female who had normal delivery during which emesis and mild cyanosis was recorded. Roentgenogram of chest shows irregular areas of consolidation in right upper lobe due to aspiration pneumonitis (arrows).

Fig. 3. Case III. A twenty-eight year old eclamptic female regurgitated during a seizure and also during anesthesia. A roentgenogram taken twenty-four hours post anesthesia shows patchy areas of increased density in both basal lung fields. This atelectatic change was due to aspiration pneumo nitis.

Chest roentgenograms during this period showed patchy areas of increased density in both basal lung fields with probable atelectatic changes (Fig. 3). There were persistent pathologic roentgenographic findings for five weeks, but these eventually cleared.

Case IV. A thirty-two year old white female was admitted with a diagnosis of cholelithiasis. On April 1, 1957 a cholecystectomy was performed. There was no note of vomiting during anesthesia. On the following day she developed cyanosis and signs of impending shock. The intern noted "labored breathing and rattling noises in her throat." She was treated as a case of pulmonary edema. During the next few days the anesthetists performed numerous tracheal aspirations. On one such aspiration a large amount of bile-stained fluid appeared without indication of vomiting or regurgitation. Also, it was noted that there was absence of the gag and laryngeal reflexes and only a weak reflex at the carina. She eventually recovered.

Roentgenograms secured during her acute episode showed patchy areas of increased density in both lung fields with atelectasis of the

Fig. 4. Case IV. A thirty-two year old female vomited during anesthesia. Postoperatively she developed some cyanosis and signs of impending shock. The bedside chest roentgenogram reveals patchy areas of increased density in both lung fields and associated atelectasis (*a*) due to aspiration pneumonitis.

medial basal segment of the right lower lobe (Fig. 4).

Case V. A twenty-eight year old Negro female gravida IV, para III, was admitted on December 18, 1956 in labor. Delivery followed within five hours and was uncomplicated, except for the fact that during the induction phase of the anesthesia she vomited large quantities of gastric contents. This was followed by considerable respiratory distress and over the next hour she gradually developed pulmonary edema. Following this the blood pressure gradually dropped and the patient developed peripheral circulatory collapse. During the next four days, tracheal aspiration was performed several times and bronchoscopy was resorted to on two occasions. However, in spite of considerable therapeutic effort she died on the fifth postpartum day.

Roentgenograms taken on December 18, 1956 revealed approximately 50 per cent collapse of the right lung with patchy areas of increased density in the left lung. On December 21, 1956 a single anteroposterior bedside roentgenogram of the chest revealed re-expansion of the right lung. There were patchy areas of increased density throughout both pulmonary fields with tendency to confluence and massive consolidation (Fig. 5 and 6).

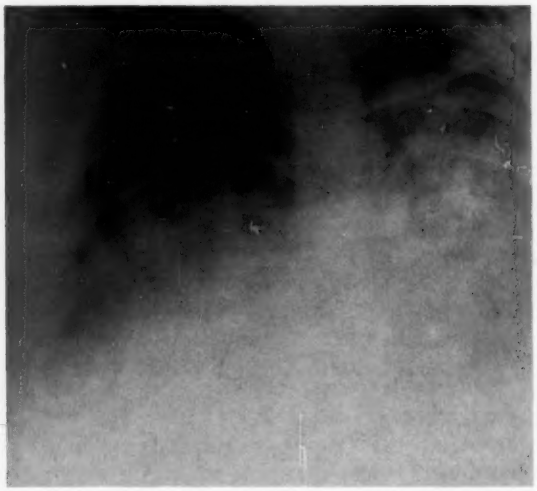

Fig. 5. Case V. A twenty-eight year old female vomited large quantities of gastric contents during delivery. Following anesthesia she developed signs of pulmonary edema and circulatory collapse. Roentgenograms made shortly after delivery show at least a 50 per cent collapse of the right lung and patchy areas of increased density in the left lung due to aspiration. Pneumothorax shown at *a*.

Case VI. A seventy-four year old white male was admitted to the Hartford Hospital on July 27, 1957 with neurologic signs pointing to

Fig. 6. Case V. Repeat roentgenogram taken three days later reveals re-expansion of the right lung but patchy confluent areas of aspiration pneumonitis throughout both lung fields persist.

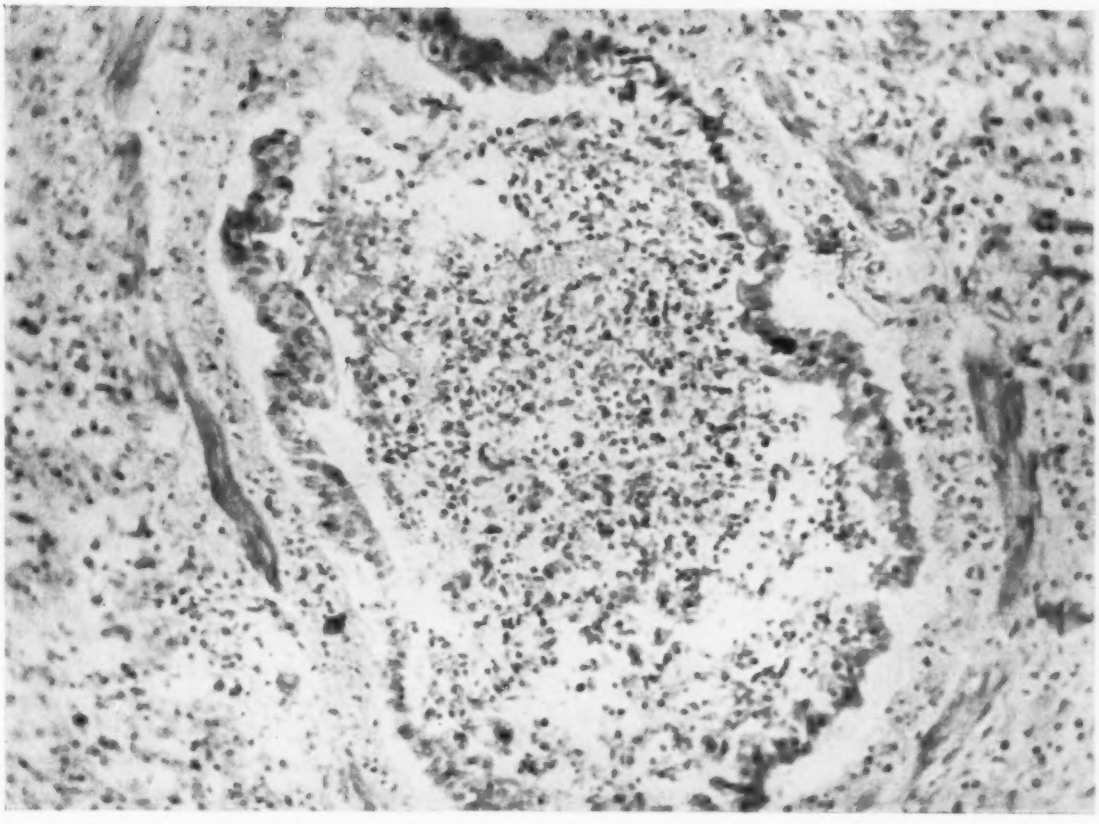

FIG. 7. Low power photomicrograph (100×) demonstrates a bronchiole containing purulent exudate and aspirated debris with reactive regeneration of bronchiolar epithelium and pneumonitic reaction of surrounding pulmonary parenchyma.

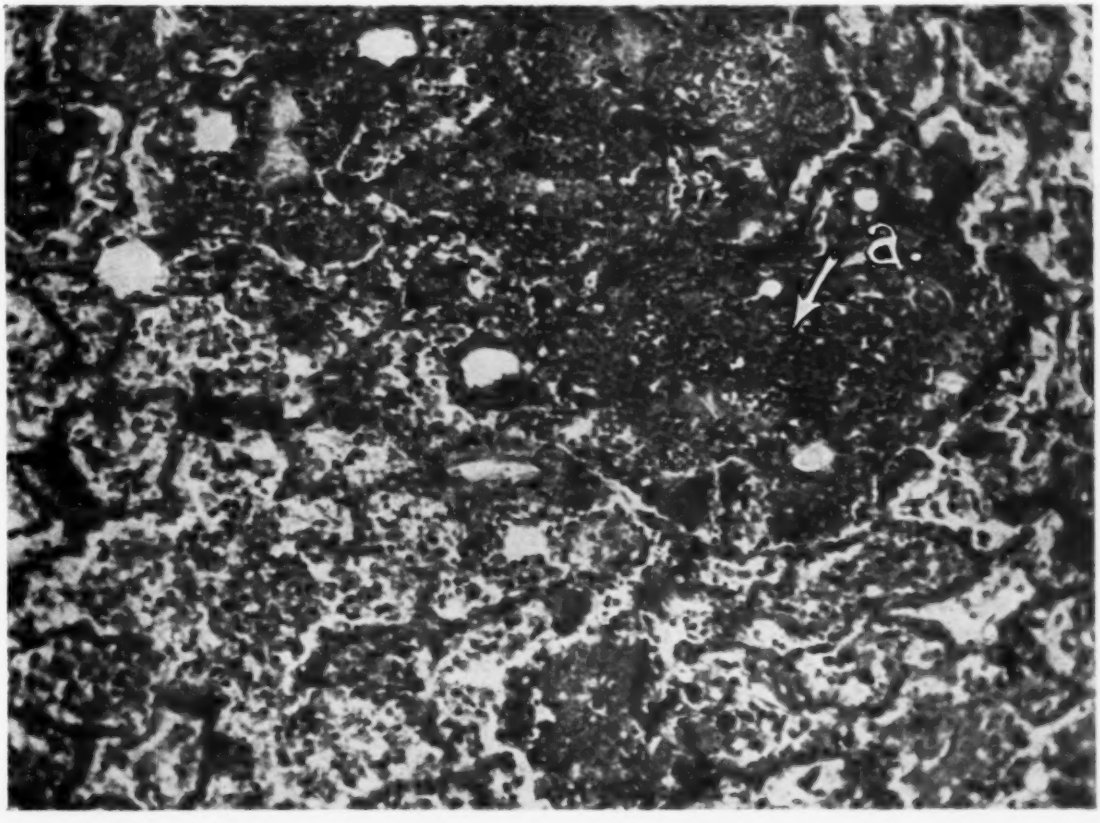

FIG. 8. Low power photomicrograph (100×) demonstrates area of pneumonitic consolidation with fibrinous and inflammatory cellular alveolar infiltrate and early micro-abscess formation at *a*.

a cerebrovascular accident in the left temporal area. Two weeks after admission a routine barium enema study revealed possible adenomatous polyps in the restosigmoid region. Repeat examination confirmed this. Since the polyps could not be reached by the sigmoidoscope, surgery was advised. Three weeks after admission a 3 cm. polyp was removed which microscopically showed carcinomatous change. Both anesthesia and operation were accomplished without incident. However, twenty-four hours postoperatively it was necessary to adjust the Levine tube. This caused violent coughing resulting in a wound dehiscence. The patient was taken to the operating room for repair of the wound under general anesthesia. During the procedure he aspirated large quantities of vomitus and required irrigation under bronchoscopy. Postoperatively the patient was incoherent and in moderate respiratory distress. The lungs were filled with moist rales and ronchi. During the next few days his condition became progressively worse. He required frequent tracheal suction. There was a spiking temperature to 104° F. Six days later a second dehiscence occurred and the patient was reoperated under general anesthesia. Following this he became increasingly dyspneic and cyanotic. There were findings of pulmonary edema. His condition gradually deteriorated and he died eleven days after the initial surgery and ten days after aspiration.

A bedside roentgenogram of the chest (Fig. 9) taken two days following aspiration showed patchy areas of consolidation throughout the right middle and lower lung fields. There were also associated atelectatic changes and mediastinal shift. Subsequent multiple roentgenograms to the time of death failed to reveal any resolution of the process which apparently was gradually progressive.

PATHOLOGY

Prior to 1940 there was practically no literature dealing with aspiration pneumonitis. Hall[3] states that he failed to find a single case report prior to 1937 after a thorough search of the literature. Since then, considerable attention has been directed to this entity, especially by anesthesiologists, obstetricians, and experimental pathologists. In 1946 Mendelson[4] reported 66 cases of aspiration pneumonitis, in 45 of which the type of aspiration was recorded; 5 aspirated solid material and 40 aspirated liquid material. He states that the solid material produces signs and symptoms of obstruction, whereas liquid material produces asthmatic wheezing, marked tachycardia, tachypnea, and cyanosis, followed in some cases by cardiac failure and pulmonary edema. Experimental studies were performed in which he introduced distilled water, normal saline, 0.1 *N* HCl, liquid vomitus, and neutralized liquid vomitus into the tracheobronchial tree of rabbits. He found that only acid caused the above described symptoms. The neutral solution produced a transient dyspnea but either minimal or no pathologic changes. This work has been repeated and confirmed. Winternitz *et al.*,[8] after insufflation of weak solution of HCl in rabbits, described an inflammatory process resembling that seen in influenza by inhalation of toxic gases. There was intense edema and congestion of the lung with hemorrhage, dilatation of the alveolar ducts and bronchioles, hyaline necrosis of the epithelium of these structures and lobular, pseudolobular and even lobar types of consolidation were encountered. Such consolidation may be serofibrinous, hemorrhagic or purulent.

Teabeaut[7] noted that if the pH of the aspirated material was below 2.5, the response was as indicated above. However, above pH 2.5 the response was similar to that found in control animals in which water was instilled. As would be expected, the type of vomitus affects the pathologic findings as well as the clinical picture. The response to macroparticulate matter is primarily granulomatous if the pH of the vomit is above 2.5. If the pH of the aspirated material is 2.5 or less, there is usually congestion, hemorrhage, de-epithelization of the bronchial mucosa and polymorphonucleosis. A combination of both types of cellular response may exist. The few pathogenic organisms present in gastric contents are not related causally to aspiration pneumonia, but may be of importance in result-

ant complications. The enzymes present in aspirated gastric contents will have little or no influence on the pathogenesis of aspiration pneumonitis or its complications.

ROENTGENOGRAPHIC ASPECTS

There is little or no mention of aspiration pneumonitis in any of the older radiologic texts except in reference to aspiration of specific substances such as oil, kerosene, etc. In more recent papers the roentgenographic findings in aspiration pneumonitis are stated to resemble those of interstitial pneumonitis. The bronchovascular markings are exaggerated and there are scattered patchy areas of increased density. Patchy emphysema and atelectasis may coexist. The lung segments most frequently involved are those which are shown experimentally to receive by natural flow the greatest quantity of fluid within the tracheobronchial tree.

Brock[2] states that the upper lobes are more frequently involved than the lower and the right lung more often than the left. He showed that the posterior and lateral aspects are more frequently involved because of the habitual posture of the patient in right dorsal and lateral decubitus. Our cases present roentgenographic findings which in general follow the pattern described above. It should be emphasized that patchy postoperative pneumonitis and atelectasis should alert one to the diagnostic possibility of chemical pneumonitis due to aspiration of gastric contents either silent or otherwise (Mendelson's syndrome).

COMMENT

Regurgitation and aspiration occur occasionally during anesthesia, immediately following recovery from anesthesia, or during the induction phase of anesthesia. Morton and Wylie[5] report that aspiration and regurgitation occur during induction twice as often as during the operation and recovery. Parker[6] states that in his experience the most serious cases resulted when regurgitation and aspiration occurred during induction. Furthermore, Berson and Adriani[1] found that in some instances the anesthetist is unaware that regurgitation and aspiration have taken place—a condition known as "silent regurgitation and aspiration."

The physician responsible for the postoperative or postpartum care of the patient is often perplexed by the development of pulmonary complications and positive roentgenographic findings in the chest. The roentgen findings, however, are nonspecific, and though aspiration pneumonitis may be suspected, it should be emphasized that definitive diagnosis is somewhat equivocal without a positive history for aspiration.

The primary pathologic changes are apparently due to the acid pH of the gastric contents. The enzymes and bacteria present are of little or no importance in the pathogenesis of this disease. However, bacteria may play a significant role in the production of secondary complications. Another etiologic mechanism in the production of such complications as abscesses, bronchiectasis, and pulmonary gangrene is obstruction of the bronchial tree by aspirated material or by the exudative parenchymal reaction caused by such material.

The 6 cases described above show considerable variation in severity and clinical course. Case II was mild, and without benefit of antibiotics underwent complete clearing within a period of three days. The other 5 were critically ill patients; 2 survived, and 3 died. Regurgitation with aspiration was readily recognized by the anesthetist in 4 of the cases, but it was silent in 2. Five patients were females. Three involved obstetric procedures, 2 were elective cholecystectomies, and one followed laparotomy for removal of an adenomatous polyp from the sigmoid. In Case V an autopsy was performed and the gross and microscopic changes were strikingly similar to those reported in experimental animals following tracheobronchial insufflation of acids (Fig. 7 and 8).

Aspiration pneumonitis, otherwise called acid aspiration syndrome of Mendelson, al-

Fig. 9. Case VI. A seventy-four year old male patient. History of aspiration of large quantities of vomitus while undergoing repair for abdominal dehiscence. Note patchy pneumonitis and atelectasis in right basal lung field with mediastinal shift from left to right.

though not new has become better understood during the last decade. Prompt preventative measures, including bronchoscopy, are not wholly successful. The roentgen appearance, though nonspecific, should be carefully weighed in postoperative cholecystectomy cases and following delivery when there is a history of regurgitation or aspiration. In many instances all modern means and methods will not prevent fatal termination of a case. By being aware of this entity and by establishing an early roentgen and clinical diagnosis, the incidence of such fatal terminations may be substantially reduced.

SUMMARY

1. A brief review of the literature dealing with aspiration pneumonitis—aspiration syndrome of Mendelson—is presented.

2. Six cases with associated roentgenographic findings are reported.

3. The roentgen aspects of the entity are briefly described.

4. An attempt is made to emphasize the importance of this lesion in patients with a history of postoperative regurgitation for any reason but particularly those following delivery and cholecystectomy. The radiologist should be alert to distinguish this form of pneumonitis from the more frequently encountered forms of patchy pneumonitis and atelectasis due to other causes.

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EXTERNAL VENTRICULAR LARYNGOCELE*

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"I have found a dilatation of the ventricle of Morgagni, up to the present unknown as it seems, which one may call 'ventricular laryngocele'. . . . " Virchow[24]

THERE have been approximately 80 cases of external ventricular laryngocele reported in the literature, with very few reports in the radiologic literature. The condition is so striking and interesting that a report of a case and a review of the pertinent literature would seem to be worthy of presentation to the radiologist.

External ventricular laryngocele is a projection of the laryngeal sacculus through the thyrohyoid membrane of the larynx, which appears as an air pouch or sac in the anterior triangle of the neck.

REPORT OF CASE

E.N., a thirty-nine year old, white male insurance broker was seen in consultation in the radiologic department of the Lawrence Memorial Hospital, with the complaint of painless swelling of the left side of the neck of three years' duration. He had first noted this swelling following hospital discharge for a perforated duodenal ulcer, three years previously. The mass at first was a pea sized, soft swelling in the left side of the neck. It gradually became larger, and became more prominent when the patient was coughing, drinking, swallowing, or exerting any other action which would increase the intralaryngeal pressure. The patient had noted that the quality of his voice had changed over the past several years, and his wife also thought that the tone of his voice had changed.

Relevant past history revealed recurrent attacks of asthma for many years, and a chronic cough, at times severe and hacking in type. He had the usual childhood diseases including whooping cough, without sequelae. The patient states that he had a weak voice all of his life, and that he was unable to shout as loud as others. On occasion he complained of a catch in his throat, which would make him lose his breath or precipitate a spell of coughing.

Following his operation for perforated duodenal ulcer, he had been on Wangensteen suction for several days, and after removal of the tube he complained of dysphagia for several weeks.

On physical examination, there was a soft fluctuant mass, 6 by 4 cm. in size, in the anterior triangle of the left neck, overlying the upper portion of the larynx. The mass was tympanitic and partially reducible. There was also a small pea sized swelling in the right side of the neck, which measured 1.2 by 1 cm. The patient had a slight huskiness to his voice and, following manual reduction of the mass, he was unable to speak until the mass had again filled with air.

Roentgenographic examination of the soft tissues of the neck showed typical findings of bilateral external ventricular laryngocele, with an internal laryngocele present on the right (Fig. 1, 2, 3 and 4).

REVIEW OF THE LITERATURE

Albucasis, a celebrated physician of the 11th century, is reported to have described a hernia or bronchocele of the anterior portion of the trachea.[13] The first description of the laryngocele was made in 1829 by Larrey,[14] the surgeon-in-chief of Napoleon's army, while in Egypt. He called these

FIG. 1. Anteroposterior view of the neck, showing the large laryngocele on the left side and the smaller laryngocele on the right side.

* From the Department of Radiology, Lawrence Memorial Hospital, Medford, Massachusetts.

FIG. 2. Lateral view of the neck with the laryngoceles superimposed and overlying the larynx.

"goitre aërien ou vésiculaire." He found that certain of the blind men, who were used as chanters of the Koran from the minarets, were disposed to this condition (Muezzin), and he also discovered the condition in several drill masters. He believed it to be the result of vocal abuse from the demand of their stentorian voices. His observations were challenged by von Hippel[25] in later years, who stated that Larrey had described either cystic goiters or dilated jugular veins, and that vocal abuse probably did not produce this, or every drill master, opera singer, or anyone who made strenuous vocal efforts would present the same condition, and the condition is quite rare.

Bennett,[2] in 1865 described the first anatomic findings of laryngocele in 17 anatomic specimens, including 9 cases of bilateral laryngocele. There have been excellent case reports by Blewett,[3] Richards,[20] Taylor,[23] Schall,[21] O'Keefe,[19] Horowitz,[9] and McLaurin,[16] to name a few.

There have been many synonyms used for the designation of this condition, a few being, laryngeal aerocele, air cysts of the larynx, monkey glands, monkey pouch, diverticulum of the larynx, and air hernia of the larynx. The term aerocele of the larynx, or laryngocele, was introduced by Virchow[24] in 1867.

The laryngeal ventricle is a normal finding in man and is found only in mammals.[23] It is a small recess, or slit, located between the true vocal folds and the false cords, and this recess ends in a blind pouch, or sacculus. The sacculus is of varying length, and if it extends higher than the upper border of the thyroid cartilage, it exceeds the upper limits of normal as defined by Virchow.[12] Embryologically,[18] it develops as an outpouching of the laryngeal cavity toward the end of the second month of intrauterine development. It is present and is of relatively large size at birth, and it commences to regress at the end of the sixth year.

It is of interest to note that the ventricle and saccule reach greatest development in the ape, orang-utang, and gorilla,[4] and may show marked degree of development extending to the thoracic cage or even to

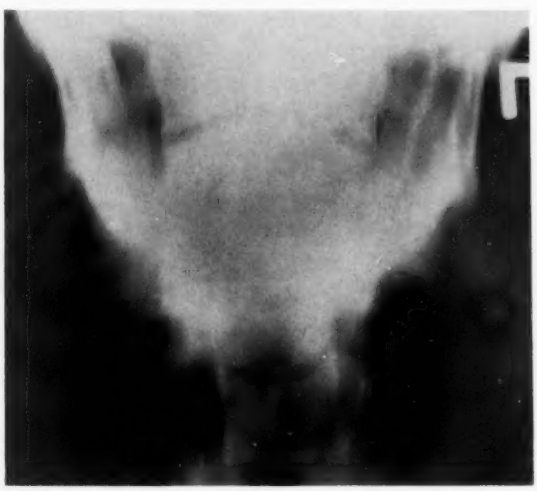

FIG. 3. Laminagram of the larynx, anteroposterior view, showing the external laryngoceles and an internal laryngocele on the right.

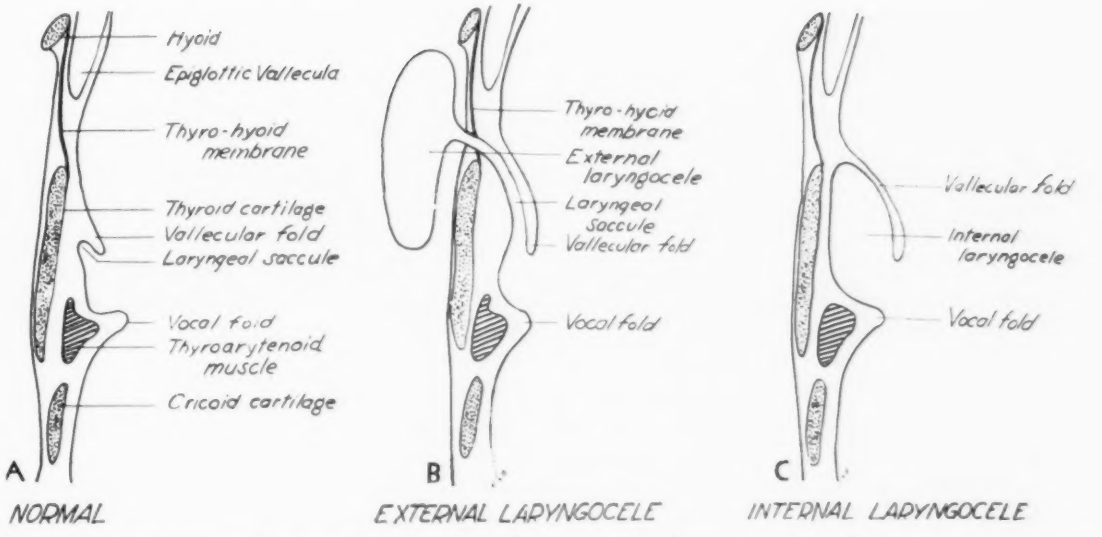

Fig. 4. Diagrammatic sections of the right half of the larynx, showing relationship of the various structures: (*A*) in the normal; (*B*) in external laryngocele; and (*C*) in internal laryngocele.

the axillae. Naturalists consider them to be a form of rebreathing apparatus.

CLASSIFICATION

Lindsay[15] has classified laryngoceles into three types: the internal laryngocele, the external laryngocele, and the combined internal and external laryngocele.

The internal laryngocele is an abnormal distention of the ventricle and the saccule, above the true cords, and extending lateral to and above the false cords. It may extend to the aryepiglottic fold, or to the base of the tongue. It may be seen as it displaces the mucous membrane deep to the false cords, and encroaches on the intralaryngeal airway.

The external laryngocele is formed when the saccule of the ventricle perforates the thyrohyoid membrane at a point of weakness, such as the point where the superior laryngeal nerve and vessels penetrate the membrane.[12] It may be found in the neck as a small cyst the size of a pea, or it may be extremely large, extending from the mandible to the clavicle. The laryngocele may be rounded, oval, or lobulated, and occasionally the neck of the cyst may be seen on the roentgenogram to extend through the thyrohyoid membrane.

The third type is a combination of the internal and external laryngocele. The voice is affected only when there is an internal type of laryngocele.

PATHOGENESIS

The condition in man may be due to predisposing, or disposing causes. The predisposing causes are the presence of an abnormally long saccule of the ventricle, which is not supported by the action of the thyroarytenoid muscle. This may be due to persistence of an infantile type of sacculus, which is, in proportion to surrounding structures, very long. It may also be due to a hernia through a congenital weakness in the thyrohyoid membrane. The condition has been described in several infants. Kan[10] has reported suffocation of a sixteen day old baby due to this condition, and Freedman[6] has observed a unilateral laryngocele in a nine week old infant.

The presence of a developmentally long sacculus subjected to the severe strain of coughing or blowing, or other similar acts would lead to the development of this condition. Some investigators[8] consider the laryngeal saccule an atavistic remnant of the air sacs of the ape, and the laryngocele the analogue of these sacs in man.

The disposing cause is an increase in intralaryngeal pressure. This may be brought about by coughing, straining, playing a wind instrument, shouting, or any act which will raise the intralaryngeal pressure. In the act of raising the intralaryngeal pressure, the true cords approximate, and the sphincter muscles about the upper opening of the larynx, the thyroarytenoid, thyroepiglottic, and aryepiglottic muscles, close. The laryngeal cavity is thus closed, and air is unable to escape. As the intratracheal pressure increases, the pressure is transmitted to the laryngeal ventricles and saccules. The true cords are less competent than the sphincters of the larynx, and the incompetence allows the full intratracheal pressure to be transmitted to the intralaryngeal structures. It may also be produced secondary to a pathologic stricture[19] at a level of the false cords, as a result of fibrosis, granuloma, or tumor.

The external laryngocele may contain fluid, or may become infected and thus give rise to pyolaryngocele. Several cases have been reported which have been associated with carcinoma of the larynx, but infection of a laryngocele is rare, and the association of carcinoma is rarer still, despite claims of an incidence of 10 to 15 per cent.[5]

DISCUSSION

The roentgen appearance of a laryngocele is so classic that according to Taylor,[23] this is "another evidence of the fact that no laryngeal examination is complete without roentgen study."

There are hardly any lesions which might create confusion in the differential diagnosis. Branchial cleft cysts, enlarged lymph nodes, or cysts and tumors of the larynx are easily eliminated. Lateral pharyngeal diverticula[11] may present a similar roentgen appearance, but are usually smaller and somewhat higher in position. A laryngocele may be associated with dysphagia,[22] as are pharyngeal diverticula. It can be readily differentiated by a swallow of barium—the pharyngeal diverticula filling, while the laryngocele, being intralaryngeal in position, does not fill. The laryngocele may cause deviation of the larynx and trachea from the midline.[9] The air cavity of the laryngocele may vary greatly in size and shape. It may contain serous fluid and a fluid level will be seen in the upright position, or it may also contain pus, which will also produce a fluid level in an upright roentgenogram. The laryngocele may be made to change in size by the ability of the patient to completely or partially distend it at will.

Lipiodol has been used to outline the laryngocele, but this procedure is quite unnecessary, as the air itself offers an excellent medium for demonstration of the cavity. Laminagraphy may be employed to great advantage to demonstrate the external and internal types, and the associated distortion of the laryngeal air passage.

SUMMARY

A case of bilateral external laryngocele is reported. This is a relatively uncommon condition and is presented because, by its characteristic appearance, it can be easily diagnosed. The historic, anatomic, and etiologic aspects are discussed.

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We wish to thank Dr. George Hanna, Radiologist, Malden Hospital, for his kindness in taking laminagrams.

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EOSINOPHILIC GRANULOMA OF THE STOMACH*

REPORT OF A CASE

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A CONFIRMED case of eosinophilic granuloma of the stomach was observed for the first time in Chile and the writers wish to call attention to this unusual lesion.

In 1937 Kaiser first described this clinical entity in a patient fifty-three years of age, who had been suffering for a period of twelve or thirteen years from gastric pains similar to those of a peptic ulcer. Later other investigators published reports on this subject.

SYMPTOMATOLOGY

Local symptoms are predominant. These include gastric pain, often periodic in its occurrence, before or after food intake; anorexia; and at times loss of weight. In certain cases, symptoms referable to the pylorus may be present, depending on the size and site of the pathologic process. Clinically, two groups of patients may be encountered: (1) Persons having had gastric symptoms for many years. They frequently present an eosinophilia in the blood and a roentgenologically demonstrable pyloroduodenal deformity. (2) Persons having had a short history of a few weeks' duration. They usually exhibit no eosinophilia and the findings of the roentgen examination are suggestive of a polyp formation of variable size.

There does not seem to be any sex or age preponderance and cases have been described in persons thirty-two to eighty years of age.

PATHOLOGIC ANATOMY

Grossly, there is a localized thickening or polyp formation in the mucosa, which is caused by the abnormal growth beginning in the submucosa. In most of the cases this formation is found in the antrum and the size varies from that of a cherry to that of a small orange. Microscopically, a granulomatous tissue with increased infiltration of eosinophilic leukocytes is seen along with fibroblasts and dilated lymphatic vessels. There is a tendency toward infiltration of the perivascular spaces. The finding of central zones of meshes of fine fibers which are relatively acellular gives the impression that prior to the development of the granulomatous process there must have been a stage of reticular proliferation. The combination of areas of different age in a given zone explains the varying histopathologic picture described by some authors.

FIG. 1. Roentgenogram showing regular outline of the stomach with elastic walls.

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ETIOLOGY

There are several theories attempting to explain the origin of these formations. Some investigators include them among the neoplastic processes (Bolck) and call them granuloblastomas; others believe them to be granulomatous in nature produced by an allergic mechanism because of the frequent association with colitis mucosa, rhinitis vasomotoria or urticaria.

ROENTGENOLOGIC FINDINGS

On reviewing the different publications relating to the roentgenologic findings, one is impressed by their diversity, depending on the size and anatomic location of the lesion. In most cases there was a filling defect of varying size in the antrum, with sharp delineation of the border—a sign which seems to be important and helpful in the differential diagnosis from other pathologic lesions in this area. In other instances there was only a deformity of the pyloroduodenal zone which usually is impossible to differentiate from a healing benign ulcer.

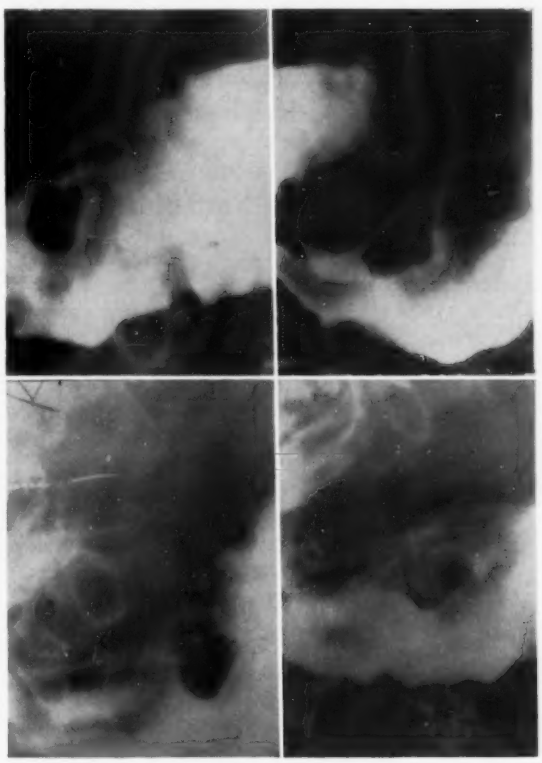

Fig. 2. Serial roentgenograms showing round filling defect, with well defined borders, in the antrum of the stomach.

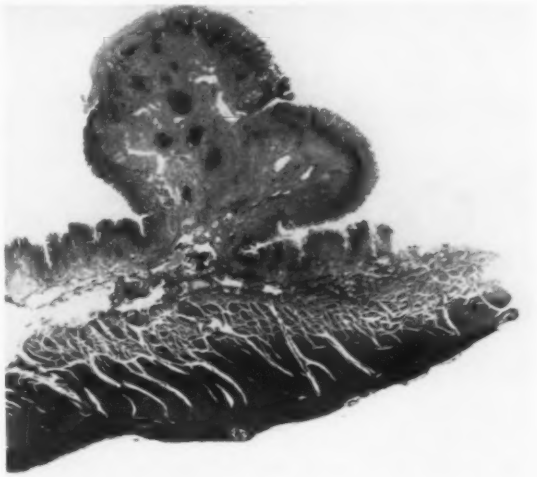

Fig. 3. Photomicrograph (×15) shows polypoid proliferation of 1 cm. in its largest diameter, united with a pedicle to the mucosa of the posterior wall of the antrum. It is almost completely covered by mucosa; a small part is ulcerated.

It is our belief that the roentgenologist should evaluate his findings by keeping in mind all the pathologic conditions which can produce a similar appearance such as benign solitary polyp, aberrant pancreas, adenomyoma and, under certain circumstances, even a malignant neoplastic process.

REPORT OF A CASE

F.O.A. White male of fifty years. 56/16051. The presenting symptoms (June 9, 1956) started about two months before admission with epigastric pain and a sensation of fullness after food intake. There had been a loss of weight of 4 pounds, with occasional vomiting during the last fifteen days.

The past history was negative. The patient drank 1 liter of wine daily until two years ago and has been a moderate smoker.

The physical examination revealed a thin man, with moderate signs of dehydration. The neck and chest were normal. Abdominal palpation produced moderate pain in the epigastrium, left hypochondrium and left iliac fossa. The liver could be felt two fingerbreadths below the costal margin and was of hardened consistency. The spleen could not be felt nor percussed. The two diagnostic possibilities con-

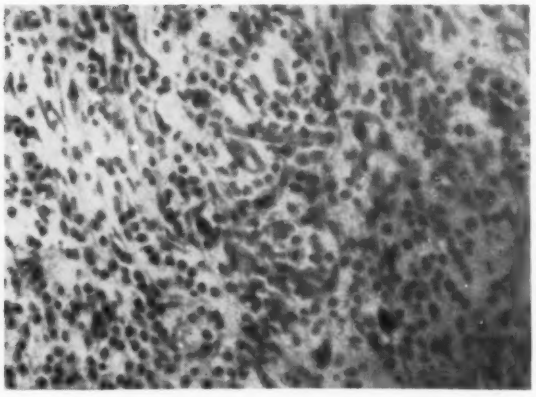

FIG. 4. Photomicrograph (×150) shows the polyp to be composed of granulomatous tissue with numerous capillaries surrounded by eosinophilic leukocytes.

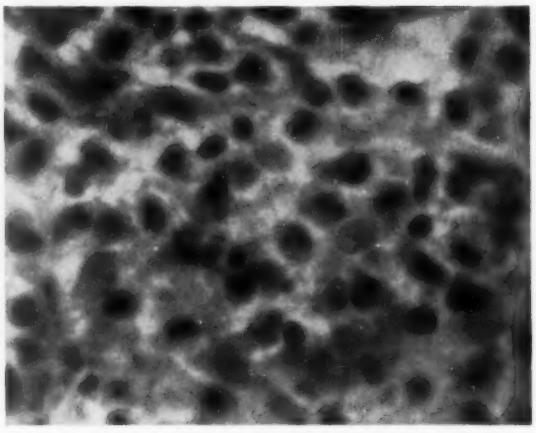

FIG. 5. Same as Figure 4 (×525). The eosinophilic leukocytes surrounding the capillaries are more clearly visible.

sidered clinically were cirrhosis of the liver and cancer of the stomach.

The examination of the gastric content revealed absence of free acid. Blood studies (nonprotein nitrogen, sugar, Kahn tests, red and white blood cell count including the eosinophil count) were normal.

The findings of the roentgenologic examination were as follows: chest, normal; esophagus, normal (no varices were found); the stomach had a regular outline (Fig. 1) and flexible walls with a normal peristalsis; in the antrum, 5 to 6 cm. from the pylorus, there was an ovoid filling defect with sharp borders (Fig. 2). The roentgen diagnosis was between solitary polyp and eosinophilic granuloma.

At operation, a small tumor was palpated in the antrum which, when the stomach was opened, had the appearance of a polyp. Not knowing the nature of this formation, a gastrectomy was performed (Hoffmeister-Finsterer type).

The gross pathologic examination revealed a portion of stomach measuring 17 cm. along the greater curvature and 9 cm. along the lesser curvature, with a smooth and glistening surface. A polyp, the size of a large pea, was found in the mucosa of the posterior wall. The mucosal folds in the antrum were thickened. Microscopically, the surface of the polyp was ulcerated and covered by granulomatous tissue. Numerous new capillaries and an inflammatory infiltration with intense eosinophilia were present. The wall contained lymphoid nodes in the center and at the periphery there was normal gastric mucosa. The mucosa of the antrum was thickened and covered with mucus. The final diagnosis was eosinophilic granuloma of the antrum (Fig. 3, 4 and 5).

Nine months after operation the patient was in good general health. He had no gastric symptomatology and had gained 8 pounds.

SUMMARY

A brief discussion of the clinical, roentgenologic and pathologic aspects of eosinophilic granuloma of the stomach is given.

A histologically confirmed case is reported in which the clinical history and roentgenologic findings suggested the diagnosis.

This case is believed to be the first one observed in Chile.

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Vol. 80, No. 1

MULTIPLE SYPHILITIC ULCERS OF THE STOMACH AND DUODENUM*

A CASE WITH UNUSUAL LUETIC MANIFESTATIONS

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IN A previous publication[1] the author surveyed the various luetic manifestations occurring in the gastrointestinal tract. The present paper concerns a patient who exhibited certain manifestations which differed from those described before.

REPORT OF A CASE

The patient is a man aged sixty-eight, who acquired lues in 1916.

During the years 1925 and 1929, his chief complaint was vomiting. In 1930 the diagnosis of tabes dorsalis was made. Since 1934 he has had aching pains in both feet and has developed ulcers (malum perforans) of the toe pads.

In 1941 melena occurred accompanied by dizziness but without dyspeptic symptoms. On admission to the hospital he was found to have urinary retention with albuminuria and pyuria. The hemoglobin value was 24 per cent. The gastric acidity was normal. Roentgenologic examination showed a duodenal ulcer. The serologic reaction was positive and the spinal fluid was normal.

During 1942 the patient was placed on a diet because of dyspepsia.

In 1948 after a traumatic fracture of the left knee, arthrodesis was carried out. The pains in the feet increased following the operation, becoming more intense and occurring within intervals of only a few seconds.

In 1950 he had a traumatic fracture of the left femur and traction was applied.

In 1952 he was admitted to a neurologic institution with the diagnosis of tabes dorsalis. The spinal fluid was still normal, and so was the electroencephalogram. The serologic reaction was positive. During the last ten years pyuria has been demonstrated on several occasions, but aside from a mild sensation of pressure during micturition he has not been in pain. The roentgenologic examination at this time showed that the left ureter was dilated and contained a calculus about the size of an almond (Fig. 1*A*). A roentgenogram of the left knee revealed that the joint had developed a total ankylosis (Fig. 2), and that considerable parts of the femoral and tibial condyles had disappeared. A roentgenogram of the left hip region demonstrated a healed, pertrochanteric fracture with enormous amounts of callus (Fig. 3).

In 1953 the patient was again admitted to the hospital because of pyuria and the ureteral calculus. He felt well and had no pain. A repeat roentgenogram (Fig. 1*B*) demonstrated an unchanged condition from Figure 1*A*. (*C*) The calculus shown in Figure 1, *A* and *B* has passed into the bladder.

FIG. 1. (*A*) Roentgenogram demonstrating a large calculus in the dilated left ureter. (*B*) Nearly unchanged condition from Figure 1*A*. (*C*) The calculus shown in Figure 1, *A* and *B* has passed into the bladder.

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Fig. 2. Roentgenogram of the tabetic left knee joint with total ankylosis following arthrodesis.

changed condition, and surgical treatment was considered unnecessary. Another roentgenogram (Fig. 1*C*) several months later disclosed that the calculus had passed spontaneously into the bladder.

In October, 1956 he was rehospitalized because of melena. He had no dyspeptic symptoms, and the gastric acidity was found to be normal. The hemoglobin value was 92 per cent and the sedimentation rate was 8 mm. The spinal fluid was normal and the serologic reaction was positive. Roentgenologic examination of the gastrointestinal tract demonstrated 3 ulcers (Fig. 4) in the greatly dilated stomach while 1 ulcer was found in the duodenum. In the stomach 1 small ulcer was seen below the cardia, 1 small ulcer in the middle of the body and 1 larger ulcer in the angular region. The latter had a diameter of about 10 mm., was penetrating and was surrounded by a fairly large vault of the mucous membrane.

The patient received penicillin and potassium iodide treatment. A later roentgenogram showed decrease in the size of the ulcers.

Survey of the antiluetic treatment: 1916,

Fig. 3. Roentgenogram of the tabetic left hip joint with healed fracture of the femur.

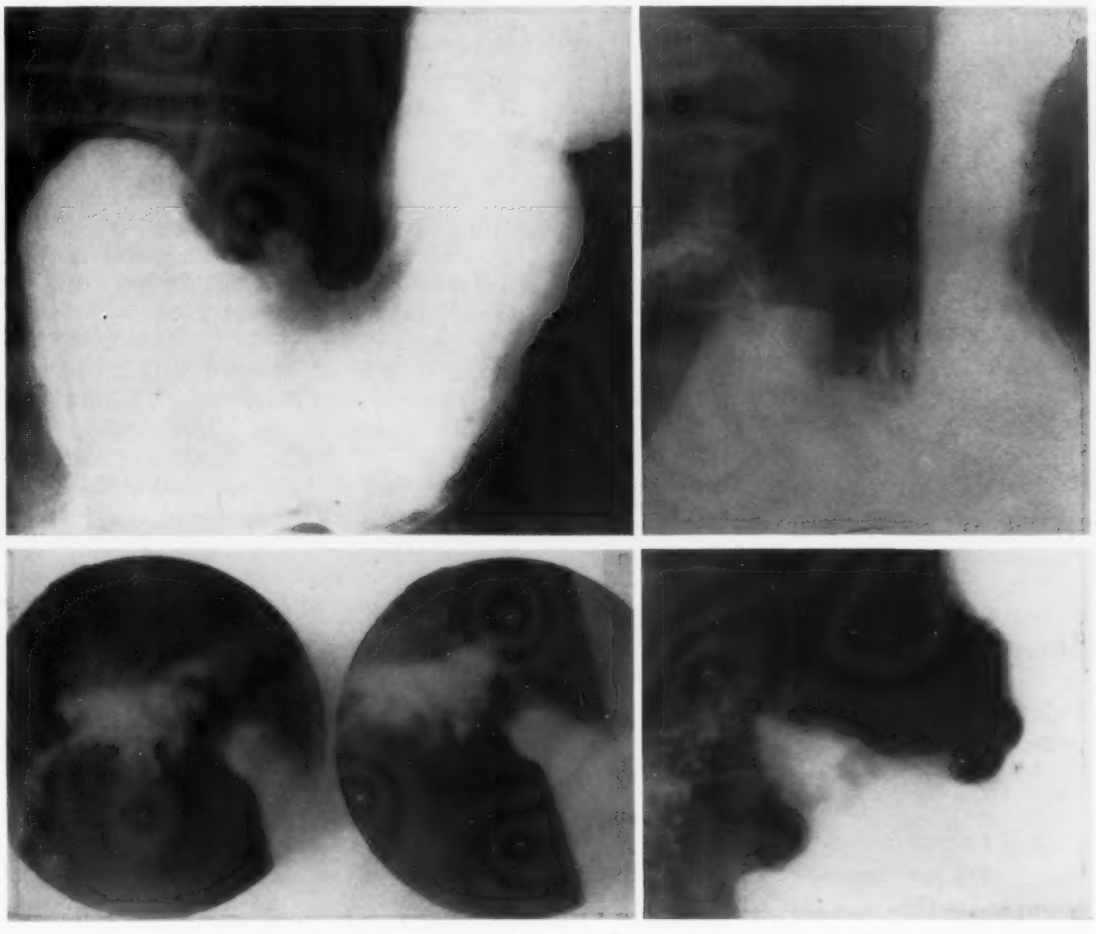

Fig. 4. Roentgenogram of multiple ulcers in the stomach and duodenum.

mercury ointment; 1930, malaria treatment; 1930–1933, 3 series of neosalvarsan and bismuth; 1952–1956, 5 series of penicillin; and 1956, potassium iodide.

Comment. The patient reported suffers from tertiary syphilis with tabes dorsalis, tabetic arthropathy and malum perforans. He has 4 gastric and duodenal ulcers but no dyspeptic symptoms. The ulcers are most likely of luetic origin.

A large ureteral calculus passed spontaneously and without pain into the bladder. The tabes dorsalis was probably responsible for the decreased sensation of pain.

SUMMARY

A case of tertiary syphilis with tabes dorsalis, arthropathy, gastroduodenal ulcers and ureteral calculus is reported.

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GAS IN THE PANCREAS AS A SIGN OF ABSCESS*

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ABSCESSES complicating infections of various abdominal organs are not at all uncommon. The presence of gas in such abscess cavities, whether it originates from the lumen of the bowel or by bacterial action, is infrequently seen. Abscesses of the pancreas may occur in association with acute pancreatitis or may follow an exacerbation of chronic pancreatitis. Surprisingly, they are very rare as a complication of a penetrating peptic ulcer. Persistence of symptoms and a constantly elevated serum amylase[9] usually indicate the development of a pancreatic abscess containing bloody, necrotic or purulent material. It may be localized within the pancreas itself or may lie chiefly in one of the "spaces" of the abdomen, such as the lesser sac or transverse mesocolon.

The occurrence of a *gas-containing* abscess, located in the pancreas, has rarely been described and a recent encounter of 2 such cases in the space of one month prompted a study of this condition and its roentgen recognition.

REPORT OF CASES

Case 1. A seventy year old female was investigated because of an upper gastrointestinal hemorrhage and symptoms suggestive of peptic ulcer. However, the causative lesion was not demonstrated by gastrointestinal barium studies.

Three years later she began to have repeated bouts of vomiting with left upper quadrant pain, and one episode of vomiting associated with diarrhea, fever and upper abdominal pain. There was increasing distention of the abdomen and soreness in the left upper quadrant.

On admission to the hospital, examination showed a seriously ill, elderly female with a respiratory rate of 40. The pulse was 150. Temperature was 100.5°F. There was dullness to percussion and diminished air entry over the left lower lobe. The liver was palpable three fingerbreadths below the costal margin. A mass was palpable in the left upper quadrant. Examination of the urine revealed a 4+ sugar with no acetone. A fasting blood sugar done three days after admission was 154 mg. per cent. The white blood cell count was 30,000.

Roentgen examination of the chest (Fig. 1) showed an elevated, fixed left leaf of diaphragm which was partially obscured by a basal pleural effusion. Beneath the left leaf of the diaphragm was a cavity with shaggy, ill-defined margins, containing an air-fluid level which extended from the lateral abdominal wall almost to the mid-line (11.0 cm.).

A supine roentgenogram of the abdomen (Fig. 2) also showed the extensive gas-containing cavity occupying the greater portion of the left upper quadrant and displacing the gas-filled stomach to the right and the splenic flexure inferiorly. The left psoas and kidney outlines were obliterated. Just to the left of the spine, superimposed on the gas shadow of the stomach, was a small cluster of radiolucencies, each measuring up to 0.8 cm. in diameter and covering a total area of 4 by 4 cm. (Fig. 3). A lateral roentgenogram (Fig. 4) showed them to be anterior to the vertebrae, within a large soft tissue mass. An intravenous urogram was normal.

A diagnosis of pancreatitis and subphrenic abscess was made on the basis of the roentgenologic findings. A small incision was made just below the left costal margin. Fluid and air gushed out through the incision and 1,500 cc. of serosanguineous, foul-smelling fluid was aspirated. The findings confirmed the roentgenologic impression of a mass associated with the pancreas. Culture of the fluid grew gram-negative lactose-fermenting bacilli sensitive to chloromycetin and streptomycin.

An empyema in the left pleural space was drained two weeks later. Injection of contrast material into a draining sinus in the left upper abdominal quadrant demonstrated a walled-off subdiaphragmatic cavity. The cluster of radiolucencies, seen on admission, was still present but reduced in size six weeks later.

One month later, a left paramedian incision

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was made over the mass in the abdomen. The omentum was adherent to the anterior abdominal wall. A plane of cleavage was found between the stomach above and the colon below. A cavity was entered and a large amount of pus was found and drained. A drainage tube was inserted. The clinical course was downhill and the patient expired one week after the final operation.

At autopsy, the peritoneal cavity contained 100 cc. of sanguineous fluid. The peritoneal surfaces were roughened and red-yellow. The splenic flexure of the colon was depressed.

The pancreas weighed 150 gm. and was edematous, especially in its tail where it showed fat necrosis with hemorrhage. A tract which had been kept open by the drainage tube was found penetrating the pancreas at the junction of middle third and tail and passing behind into the spleen and retroperitoneal structures. A continuation of the sinus tract from the pancreas had caused a 3.5 cm. hole in the lower portion of the spleen. The spleen was enlarged and weighed 665 gm. Its surface was roughened by fibrous adhesions which bound it to the pancreas and diaphragm.

Fig. 1. Case 1. Left subphrenic abscess with air fluid level, elevated (and immobile) left leaf of diaphragm and left pleural effusion.

Fig. 2. Case 1. Large collection of gas in shaggy irregular left subphrenic cavity. Stomach displaced to the right and downward. Splenic flexure displaced downward. Collection of gas bubbles is seen in abscess to the left of third lumbar vertebra and superimposed on stomach shadow (see Fig. 3).

Microscopically, the pancreas showed areas of fibrosis and collections of chronic inflammatory cells. The final pathologic diagnosis was pancreatitis, complicated by pancreatic and left subphrenic abscesses.

Case II. A forty-four year old white female was admitted to the hospital with the chief complaint of a right upper quadrant mass. A year prior to admission, the patient had right upper quadrant pain associated with mild jaundice which gradually subsided over a period of three weeks. Six weeks prior to admission, she experienced sudden onset of right upper quadrant pain which went through to the back and was associated with distention of the abdomen. This was followed by jaundice which lasted for two weeks and gradually subsided.

Examination revealed a thin woman with a protuberant abdomen. On the right side was a mass which moved with respiration, was fluctuant on palpation and tender on deep pressure. There was a suggestion of tympany to the mass and shifting dullness in the abdomen. The tem-

Fig. 3. Case 1. Enlarged view of cluster of gas bubbles shown in Figure 2.

perature was 100.6°F. The pulse was 95. The respirations were 33. The sedimentation rate was 95. The leukocyte count and the serum bilirubin were within normal limits.

A plain roentgenogram of the abdomen (Fig. 5) showed a collection of small radiolucencies, up to 0.8 cm. in diameter, located to the right of the spine, extending to the lower lumbar region, and covering an area 9 by 10 cm. Small areas of calcification were present in the left upper quadrant. The psoas shadow was preserved, but the lower pole of the right kidney was not demonstrated. A cholecystogram did not demonstrate the gallbladder. A barium enema study (Fig. 6) showed that the radiolucencies were contained within a mass which depressed the proximal transverse colon. An upper gastrointestinal examination (Fig. 7) revealed an extrinsic defect on the greater curvature of the gastric antrum and duodenal cap. The duodenal cap was deformed. Only a small amount of barium was demonstrated in the duodenal loop. Its mucosal pattern appeared flattened and, in some places, edematous.

At operation a mass was found in the mesocolon in front of the duodenum and pancreas. There was fat necrosis and the pancreas contained calcific plaques in the tail and body. The mass was incised and a quantity of gray-yellow purulent material was evacuated, together with large pieces of necrotic pancreatic material. The abscess was drained by a large tube. A roentgenogram of the necrotic fragments of material failed to show any radiolucencies similar to those seen on the previous roentgenograms.

The material from the abscess was like putty and varied in color from gray-white to black. It was generally soft but the lighter areas were firm. Lining part of this mass of tissue, there was a gray-brown, smooth membrane which appeared to represent the wall of a cyst. Microscopic examination revealed the presence of acute inflammatory cells, masses of necrotic debris in which the ghost-like remnants of altered fat cells and masses of calcium soaps and fatty acid crystals could be identified. Considerable hemorrhage was present. The pathologic diagnosis was pancreatitis with fat necrosis and pseudocyst. A culture of the material grew Streptococcus viridans and occasional Clostridium welchii.

Postoperatively, a gastrointestinal series showed a deformed duodenal cap. A fistulous tract, 1 cm. in length (Fig. 8), passed inferiorly from the duodenal cap into a triangular cavity,

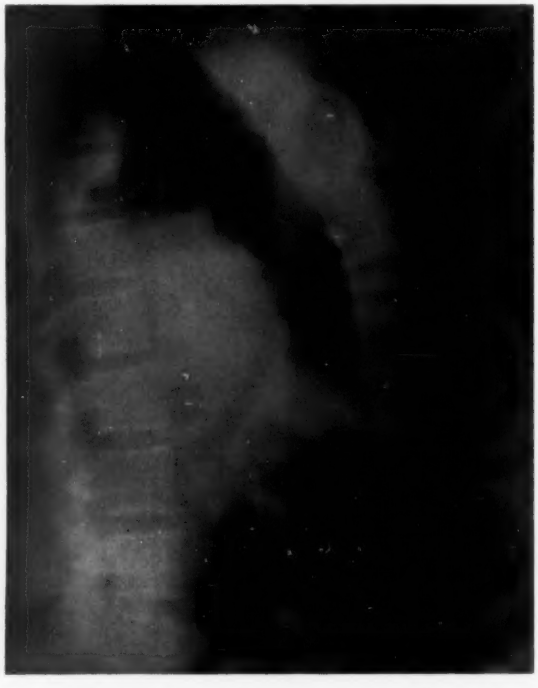

Fig. 4. Case 1. Lateral roentgenogram shows large subphrenic collection of gas, a mass anterior to the bodies of T12, L1, L2 and L3 and the cluster of gas bubbles anterior to L3.

measuring 3 by 2 cm. The remaining duodenum was normal.

REVIEW OF LITERATURE

In most of the previously reported abscesses related to pancreatitis, the chief roentgen finding was the presence of a large cavity with a long air-fluid level, similar to that in Case 1. In 1913 Bittorf[2] described 2 cases of pancreatic abscess with roentgenographically demonstrable gas. Abdominal roentgenograms in the first case showed a large clear air space in the upper abdomen under the elevated left hemidiaphragm. The gas extended to the right of the midline, being separated from the right hemidiaphragm by the liver. At operation, a gas-containing abscess was found in the lesser sac, originating from a gangrenous pancreas. Roentgenograms in the second case showed elevation of the entire diaphragm, more marked on the left side. The stomach bubble was absent, being replaced by a large gas shadow. At operation, a large abscess containing necrotic fragments of pancreas was found in the lesser sac.

FIG. 6. Case 11. Cluster of gas vesicles in abscess which is causing depression of proximal transverse colon.

FIG. 5. Case 11. Collection of gas bubbles to right of L_3 and L_4, obscuring lower pole of right kidney. (Dense shadows represent old barium in cecum and rectum.)

In 1950 Samuel[10] described a pancreatic abscess which was situated behind the stomach in the lesser sac, containing an air-fluid level approximately 7 cm. long. Lateral roentgenograms showed the stomach displaced forward and stretched around the abscess cavity. Buckstein[3] described a huge gas-containing pancreatic abscess in which the air-fluid level was equivalent to the width of four vertebral bodies in the upright position and which extended from the seventh to the twelfth thoracic vertebrae in the lateral decubitus position. Schinz[12] illustrated a case of abscess in the tail of the pancreas, containing a single gas-filled cavity, several centimeters in diameter.

Less frequently described are instances of pancreatitis with abscess formation characterized by small collections of gas. In 1949 there were recorded 2 autopsied cases of acute pancreatitis.[4,5] The first resulted in a pancreatic abscess which perforated into the transverse colon and ex-

Fig. 7. Case II. Oblique roentgenogram, showing gas vesicles contained in abscess which is deforming greater curvature of gastric antrum and duodenal cap.

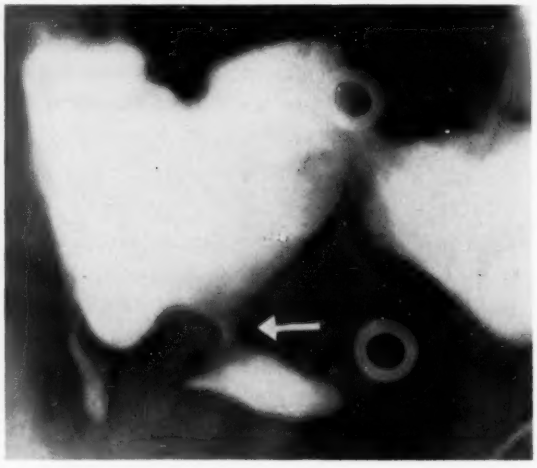

Fig. 8. Case II. Spot roentgenogram of duodenal cap, obtained two months after operation, shows barium passing vertically downward through a fistulous tract (arrow) and entering a cavity in the head of the pancreas. Barium is just entering the second portion of the duodenum.

tended up under the left leaf of the diaphragm. Roentgen studies showed a cluster of radiolucencies, having the appearance of gas bubbles, adjacent to the under surface of the diaphragm. There was plate-like atelectasis in the left lung base. It could be presumed that, in this case, the gas in the abscess originated from the transverse colon. In the second case, the abscess extended down both paraspinal gutters and caused narrowing of the sigmoid colon. Roentgenograms showed a "peculiar gas pattern in the right lower abdomen." A gastrointestinal series showed a "wild pattern of jejunal and ileal mucosa."

Few comments are made about the bacteriology of pancreatic abscesses. In a case of chronic pancreatic abscess, reported by Beller and Nach,[1] Staphylococcus aureus was cultured. In another case,[4] colon bacilli and staphylococci were grown.

DISCUSSION

Abscesses associated with pancreatitis may remain within the substance of the pancreas and are, therefore, likely to be small or only moderate in size. Others extend beyond the confines of the pancreas to nearby sites (Case II) or more distantly (Case I). They have been described as perforating into the stomach,[7] the duodenum,[13] and the colon.[4] They may extend directly or via such organs to form fistulous communications with the skin surface. In addition to the commonly described signs seen in association with pancreatitis and pancreatic abscess, such as obliteration of organ outlines, calcifications, sentinel loop of distended small bowel, distorted duodenal mucosal pattern, extrinsic pressure defects on stomach and duodenum, the presence of gas in the abscess cavity must always be looked for. In those cases with communication of the abscess with stomach, duodenum or colon, the presence of gas is easy to explain. In others, it must be concluded that gas-forming organisms are present[8] or that an undiscovered communication exists.

Apparently the most common direction of spread from the pancreas is into the lesser peritoneal sac which lies immediately anterior to the pancreas. Once in this large space, the inflammation may spread widely and produce a large mass, containing purulent material and necrotic fragments of

pancreas and, on occasion, gas. In these instances, a long air-fluid level, indicating the diameter of the abscess cavity, may be seen on roentgenograms when horizontal beam techniques are used.

When gas is present in that part of an abscess still within the confines of the pancreas or is in an abscess which is in a confined space, such as retroperitoneal tissues,[5] or when it contains numerous fragments of necrotic tissue not yet liquified, it is less likely to be seen as a unilocular cavity with a single air-fluid level. Under these circumstances the gas is more likely to be dispersed among the solid material and to produce a mottled appearance. This appearance is not diagnostic of pancreatic abscess and may be seen in abscesses in other viscera or connective tissues.[8]

When first seen, the gas is present as small, spherical, radiolucent bubbles. The amount of gas may increase and the bubbles enlarge and coalesce. In later stages single or multiple fluid levels may appear and, eventually, the trapped gas may reveal the shaggy roof of the walled-off abscess.[8] Holmes and Robbins[6] state that the radiolucencies which are sometimes seen in cases of pancreatic necrosis are not gas but are areas of partially necrosed fat. In Case II, the roentgenographic study of the semisolid contents of the abscess, after operation, failed to show the radiolucencies demonstrated preoperatively. It is reasoned that if, among the fragments of necrotic pancreas, blood and exudate, there had been globules of partially necrosed fat sufficiently large to produce the bubble-like radiolucencies shown on the roentgenograms, they would still have been present among the debris after evacuation of the abscess cavity. However, surgical drainage of an abscess would allow any trapped gas to escape and not be visible in the roentgenogram of the removed material.

The pattern of small, entrapped bubbles of gas can be present in bulky masses of fecal material in the transverse colon or in masses of ingested food material in the stomach, and must be differentiated. Similarly, gas vesicles can occur in abscesses of renal, perirenal, pericholecystic and retroperitoneal regions, as well as the abdominal wall.[11] These could, by their location near the pancreas, be mistaken for pancreatic abscess. However, the presence of an associated subphrenic abscess favors the diagnosis of a pancreatic lesion. Small gas vesicles in pneumatosis cystoides intestinalis may resemble those seen in pancreatic abscess.

Small air-fluid levels in diverticula about the duodenum or jejunum must be distinguished from those occurring in pancreatic abscess.[10] It must be borne in mind that a gas-containing abscess in almost any location in the abdomen may have originated in the pancreas and an investigation of such findings with this in mind should be instigated. Once well established, an infected pancreatic pseudocyst can be considered, for all practical purposes, as identical to a pancreatic abscess.

SUMMARY

1. Two cases of gas-containing pancreatic abscess are added to those described in the literature. Both exhibited roentgenologically demonstrable clusters of small (0.1 to 0.8 cm.) radiolucent gas bubbles in or near the region of the pancreas. The size of the entire collection of gas bubbles was 4 by 4 cm. in one case, and 9 by 10 cm. in the other.

2. Extension of the inflammatory process beyond the usual position of the pancreas was seen in both cases. In one case there was a left subphrenic abscess with associated pleural effusion and paralysis of the diaphragm. In the other case, the collection of radiolucent gas bubbles extended to the lower abdomen.

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ADDENDUM

Three additional cases of gas-containing pancreatic abscess have been encountered since the preparation of this paper. Of the total of 5 cases, 4 showed collections of gas

bubbles on the roentgenograms (one showed a single cavity with irregular walls); 3 were fatal. The contention that an abscess or pseudocyst complicating pancreatitis can be predicted from a persistently elevated serum amylase is unfounded, since in 2 cases in which serial amylase determinations were carried out, the levels were normal.

In a series of 6 cases of gas-containing pancreatic abscess, reported by Felson (*J.A.M.A.*, 1957, *163*, 637–641), all showed multiple radiolucencies in the region of the pancreas; 5 had hyperglycemia (only one of our cases had hyperglycemia); and 4 died. In one case, gas bubbles were seen in the abscess at the time of operation. In both, Felson's series and ours, the diagnosis of pancreatic abscess was made roentgenologically in a substantial proportion of cases.

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ACUTE PANCREATITIS WITH PERITONEAL FAT NECROSIS—ROENTGEN DIAGNOSIS*

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THE accurate diagnosis of acute pancreatitis requires coordination of the clinical and laboratory findings. Elevation of the serum amylase is important as are other laboratory tests when positive, although there are circumstances in which the serum amylase may be normal in the presence of acute pancreatitis.

The roentgen method of diagnosis of pancreatitis has steadily increased in importance as new signs have been recognized. The roentgen signs alone may not be specific but may be extremely helpful when evaluated with clinical findings.

A method of direct visualization of the pancreas following cholecystectomy and insertion of a T tube in the common bile duct has been described by Doubilet, Poppel and Mulholland.[3] A polyvinyl catheter about 50 cm. long is inserted into the main pancreatic duct for a distance of 4 or 5 cm. and then brought out through the duodenum and common bile duct along the T tube. A pancreatic fistula is thus produced which can be controlled for weeks or months and closed by removal of the tube. Injection of 70 per cent diodrast produced a diffusion of the contrast medium throughout the parenchyma due to the destruction of connective tissue in pancreatitis. In the normal pancreas only the ducts are visualized.

Further studies of the pancreas by means of pancreatography have been reported more recently by Doubilet and Mulholland.[4] These were carried out in conjunction with the therapeutic procedures of sphincterotomy and drainage of the pancreas by plastic tube inserted into the pancreatic duct. Observations on the pathologic condition of the pancreas could be made as well as variations in the ductal system of the gland.

An intravenous contrast medium for direct roentgen visualization of the pancreas has not yet been produced. Until this method is developed, reliance on indirect signs must be made.

The roentgen signs in acute pancreatitis have been described in considerable detail by Poppel in Roentgen Manifestations of Pancreatic Diseases.[5] The importance of functional changes in the adjacent structures such as spasm of the stomach, duodenum, and small and large bowel, followed later by ileus has been emphasized. Edema of the mucosa of these organs may also occur. These findings are more evident if barium is given orally, but the appearance of localized gaseous distention affecting the duodenum, and the small and large bowel on the plain film of the abdomen has been described by Barry.[1] The "papillary sign" has also been noted by Poppel[6] representing edema of the papilla of the ampulla of Vater in the second portion of the duodenum.

The pancreas may be tender on fluoroscopic study and there may be generalized enlargement of the structure itself. Tumefactions in the parapancreatic area are often seen and usually are due to the formation of accumulated fluid or pseudocysts. Location of the pseudocysts is most common in the retroperitoneal areas, the lesser omental sac, the gastrocolic ligament and the subphrenic regions.

There is a common association of biliary tract disease and pancreatitis due to the close anatomic relationship of the bile ducts and pancreatic ducts. It is likely that there are many unsuspected cases of pancreatitis which have caused the predominant symp-

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toms in what were thought to be rather severe cases of cholecystitis. The roentgen demonstration of calcified biliary stones may point toward this relationship in a given case.

The presence of basal pneumonitis or atelectasis has been described frequently and this may be accompanied by pleural effusion. There may be associated elevation and limitation of motion of one or both leaves of the diaphragm.

The pulmonary and pleural changes are considered to be due to chemical irritation or infection transmitted by the lymphatics through the diaphragm from the abdomen or to direct fistulous communication.

There are general manifestations in the abdomen somewhat similar to those occurring in generalized peritonitis. Generalized increase in density throughout the abdomen may be due to fluid formation. There may also be loss in normal outline of the organs such as the kidneys and the psoas muscle shadows. The peritoneal fat layer may also be obscured.

The roentgen demonstration of lipoid necrosis in the peritoneal fat tissues has been mentioned in only 1 case in the literature by Baylin and Weeks.[2] It is the purpose of this paper to report 2 cases in which peritoneal fat necrosis can be identified similar to that reported by Baylin and Weeks.[2] The important finding is that of mottled areas of increased density of 1–3 cm. in diameter scattered throughout the abdomen and representing fat necrosis with saponification.

REPORT OF CASES

Case 1. Mrs. V.F., aged fifty-six, a large obese individual, was admitted to St. Lucas Hospital, Faribault, Minnesota, on January 24, 1952 after the sudden onset of severe pain twenty-four hours previously in the right upper quadrant and epigastrium. Her physician had administered morphine before admission without relief of pain.

The abdomen was soft although tender to palpation in the epigastrium. No masses were palpable. The pulse was weak but regular with a rate of 76. The blood pressure was 110/60 mm. of Hg. Temperature was 97.6° and the respiration rate was 24 per minute.

She had been in good health until the previous January 1, 1952 when she ate a heavy meal including considerable baked beans and pork. Since that time, she had intermittent gas pains.

Laboratory studies on admission showed the urine to have 1 plus albumin, but otherwise negative. Her white blood cell count was 28,000 with a differential count of 84 per cent polymorphonuclear neutrophil leukocytes. The clinical impression was acute cholecystitis and cholelithiasis and it was decided to treat her conservatively.

By January 28, 1952, the fifth hospital day, the patient had shown moderate improvement, objectively. There was no localized pain. Diarrhea was present. The temperature was elevated to 99.2°F. The first roentgenograms of the abdomen on this date indicated multiple variable densities scattered throughout the abdomen. Etiology of the densities could not be accurately determined, although the possibility of implants from some unknown primary abdominal tumor was considered. There was slight gaseous distention of several loops of small and large bowel thought to be due to paralytic ileus. On January 30, 1952 the white blood cell count was 27,900. On February 1, 1952 a round mass could be palpated in the mid-line above the umbilicus. A chest roentgenogram revealed bilateral basal pneumonitis and pleural effusion. A cholecystogram on February 4, 1952 indicated a nonfunctioning gallbladder. There were noted very prominently in the right upper quadrant, multiple nodular densities of unusual appearance which later proved to be due to fat necrosis (Fig. 1).

During the next few days, the patient's condition rapidly deteriorated. The temperature remained elevated to 99–101°F. and she became more toxic. An upper gastrointestinal study on February 6, 1952 (Fig. 2) showed a mass posterior to the stomach, deforming and displacing it. There also appeared to be a mass inferior to the stomach which was considered to be due to a tumor or cyst of the pancreas or kidney. On February 8, 1952 an intravenous pyelogram revealed displacement of a normal appearing left kidney inferiorly by a retroperitoneal mass thought to be either in the pancreas or adjacent areas. A chest roentgenogram on February 11, 1952 (Fig. 3) indicated increasing pleural effusion bilaterally. On the same day a barium enema examination showed displacement of the transverse colon by a large mass in the right epigastrium which was assumed to be the gallbladder markedly enlarged.

Because of the declining condition of the patient and lack of response to other therapy, it was decided to do an exploratory laparotomy on February 12, 1952. The omentum was covered with multiple necrotic areas. The bowel was adherent everywhere. The liver appeared normal and the gallbladder was only slightly dilated. The pancreas was edematous and contained areas of necrosis, typical of acute pancreatitis. A large necrotic abscess was drained through the omentum from the lesser omental sac. Postoperatively, the patient was in shock and in spite of all attempts to stimulate her, she expired in the evening of the day of the surgery. No autopsy was obtained. A biopsy of the omentum showed fat necrosis with leukocytic infiltration considered to be secondary to acute pancreatitis.

Case II. Mrs. E.S., aged sixty-six, was admitted to the Owatonna City Hospital, Owatonna, Minnesota, on November 20, 1952, with severe pain in the periumbilical area associated with nausea and vomiting. The pain radiated to the back in the upper lumbar region and had been present for three hours. Morphine sulfate gr. 1/3 was given with moderate relief. The blood pressure was 128/82 and the pulse rate 82. The urinalysis was essentially negative. The white blood cell count was 16,600, the red blood cell count 4,920,000 and hemoglobin was 15.3 gm. The patient had had one episode of illness about thirty years previously when she had pain, chills, fever, and jaundice. She was told she had "ulcers" at that time.

During the following forty-eight hours, the pain continued and the patient had frequent emeses. The abdomen became increasingly distended. The bowel sounds were faint but audible. Diffuse, generalized tenderness was present over the abdomen but more marked in the epigastrium. No definite palpable mass was present. Roentgenograms of the abdomen (Fig. 4) on the day of admission revealed gallstones in the right upper quadrant but otherwise no significant abnormalities. The chest was negative.

Fig. 1. Case I. February 4, 1952. Attempted cholecystogram showed nonfunctioning gallbladder. The multiple nodular densities of mottled character are visible in the gallbladder region. These densities represent the fat necrosis in the area.

Fig. 2. Case I. February 6, 1952. Upper gastrointestinal study indicated displacement of the stomach by an adjacent mass, representing a pseudocyst in the lesser omental space. There are also multiple nodular densities throughout the abdomen representing fat necrosis. The left kidney is displaced inferiorly by the pseudocyst.

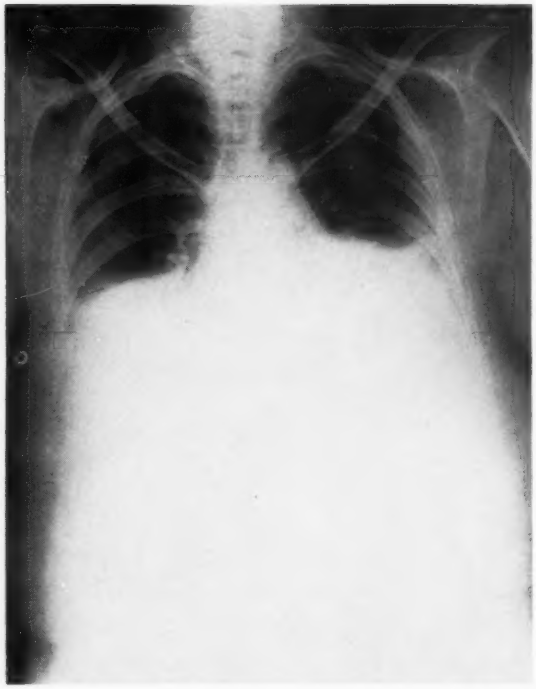

Fig. 3. Case I. February 11, 1952. Bilateral pleural effusion and evidence of basal pneumonia and atelectasis—a frequent complication in acute pancreatitis.

On November 22, 1952, forty-eight hours after the onset of the illness, the temperature was 103.8°F. and the pulse rate 135. The white blood cell count was then 12,500. Roentgenograms of the abdomen (Fig. 5) revealed the probable presence of fluid and there were noted multiple nodular densities throughout the abdomen. The possibility of these densities being due to fat necrosis was considered, although clinically the diagnosis of cholecystitis and cholelithiasis with some unusual complication appeared to be the most plausible. There was no appreciable bowel distention and no masses were visible at this time. An exploratory laparotomy was done on the same day. The abdomen contained bloody fluid and there were many areas of fat necrosis. The gallbladder was markedly distended. The common bile duct was explored and a catheter could be readily passed into the duodenum after the ampulla was dilated with a metal sound. After placing a T tube in the common duct, the gallbladder was removed. The lesser omental cavity was opened and considerable bloody fluid evacuated. The pancreas was distended and edematous along its entire length. A Penrose drain was placed in the lesser omental cavity and brought out through a stab wound. The pathology report indicated several gallstones in the presence of chronic cholecystitis.

Postoperatively, the patient continued to be toxic with the temperature and pulse remaining elevated. The respiration rate was also increased to 25 or more per minute. A chest roentgenogram on December 3, 1952 (Fig. 6), sixteen days after the onset, revealed areas of pneumonitis in the base of the left lung and pleural effusion on this side. A repeat chest roentgenogram on December 24, 1952 showed persistent pulmonary lesions. On December 30, 1952 an abdominal mass could be palpated in the midline. The mass was firm and did not move with respirations. This was assumed to be a pseudocyst of the pancreas. Roentgen-ray studies of the stomach (Fig. 7) with barium showed pressure on the stomach from a retroperitoneal mass and there were changes in the duodenum which were thought to be secondary to pancreatitis. By January 24, 1953, the sixty-fifth hospital day, the patient felt moderately im-

Fig. 4. Case II. November 20, 1952. Calcified gallstones in the right upper quadrant. Normal fat structures outlining the kidneys, liver, and lateral peritoneal spaces. Psoas muscle shadows are very distinct.

Fig. 5. Case 11. November 22, 1952. Fat necrosis evidenced by mottled, variable densities throughout abdomen. The kidneys and liver are partially obscured and psoas muscle shadows are indistinct.

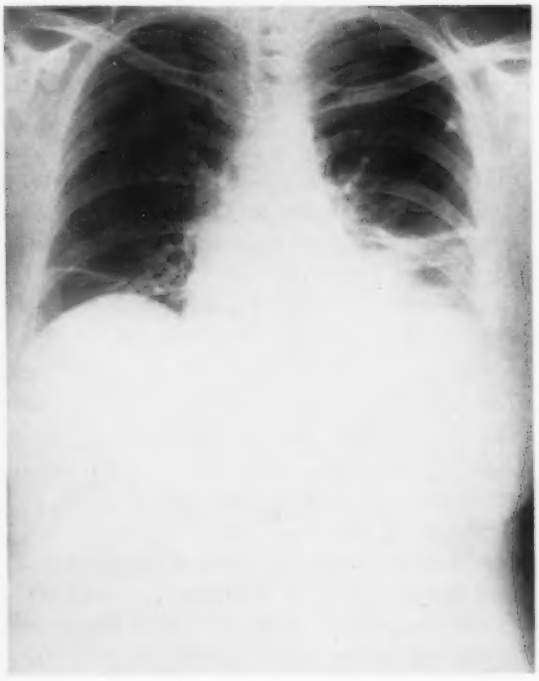

Fig. 6. Case 11. December 3, 1952. Chest roentgenogram indicating pneumonitis bilaterally, more marked on the left side. There is slight pleural reaction on the left side.

proved, although still having a daily temperature of 100°F. with a pulse rate of 104 and a respiration rate of 26 per minute. Drainage had persisted from the abdominal wound where the lesser omental cavity had been opened. The patient was discharged to her home.

On January 30, 1953 hematemesis of about one quart of blood occurred and she was readmitted to the hospital. Transfusions were given but the patient's condition rapidly declined and after repeated hematemeses, she expired on February 4, 1953, the seventy-seventh day after the onset of the illness. No autopsy was obtained.

DISCUSSION

Severe hemorrhage is a fairly common complication in pancreatitis and according

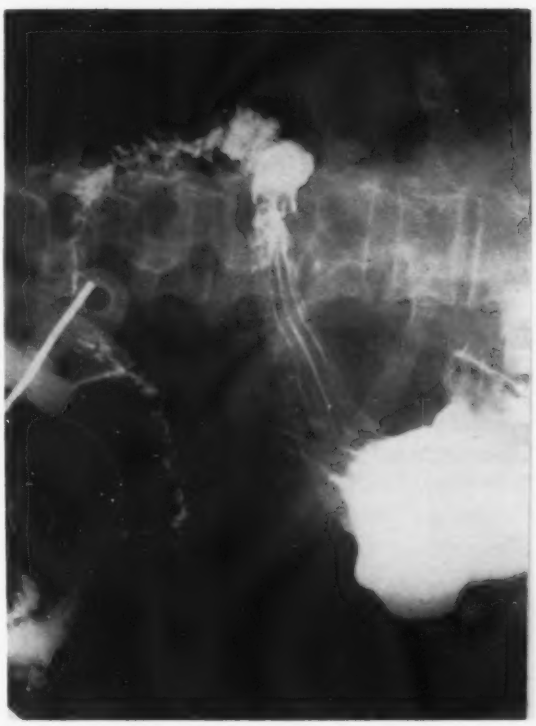

Fig. 7. Case 11. December 30, 1952. Gastrointestinal study showing deformity of the stomach due to extrinsic compression from a retroperitoneal mass, representing a pseudocyst of the pancreas in the lesser omental sac.

to Doubilet and Mulholland is probably due to high content of trypsin in the circulation causing changes in the clotting mechanism.

There are many areas in the peritoneal cavity and adjacent tissues which contain considerable amounts of fat. A common and well known observation in the roentgenogram is the lesser density produced by the fat areas in contrast to more solid structures or fluid or vascular structures. The kidneys are clearly outlined by fat layers as are the shadows of the psoas muscles and muscles in the lateral abdominal wall. There is a considerable amount of fat in the omentum and mesentery and in the infrahepatic areas.

In the presence of severe pancreatitis, obstruction of the pancreatic duct frequently is present with resulting extrusion of excess pancreatic enzymes into the pancreatic tissues. This produces marked destruction and necrosis of the pancreas with release of enzymes into the peritoneal cavity.

The pancreatic enzymes rapidly transform the fatty tissues into calcium soaps by combination with calcium from the blood serum. This is proved by a common laboratory test of determination of serum calcium. If the calcium is below 7 mg. per cent, prognosis of fatal termination of the case can usually be given.

It is apparently the greater density of these islands of calcium soaps in necrotic areas contrasted with the more normal areas of unaltered lipoid material which produces the nodular appearing shadows seen in the roentgenogram.

The identification of this finding may be of great importance in the diagnosis of pancreatitis when not already made by laboratory study. It indicates a very serious prognosis in those cases in which the diagnosis is already known.

The severity of the disease when the findings of lipoid necrosis are present on the roentgenogram suggests that many of the other complications of pancreatitis are likely to occur in a given case. Both cases reported developed hemorrhagic peritoneal fluid, pseudocysts and inflammatory lesions in the lungs and pleura.

The indication of lipoid necrosis may be present in a fairly short time as demonstrated in a roentgenogram at forty-eight hours following a normal roentgenogram taken several hours after the onset of the disease. This usually constitutes evidence of the very fulminating nature of this type of pancreatitis.

SUMMARY

Two cases of severe acute pancreatic necrosis have been reported in which the presence of peritoneal lipoid necrosis was demonstrable on the roentgenogram.

Both cases terminated fatally following development of numerous complications including pseudocysts, hemorrhagic peritoneal fluid, and pulmonary and pleural lesions.

Observation of the rather typical nodular lesions of fat necrosis in the peritoneal cavity may assist in the roentgen diagnosis of acute necrotic pancreatitis.

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VOL. 80, NO. 1

INFLAMMATORY PSEUDOPOLYPOSIS OF THE SMALL AND LARGE INTESTINES WITH THE PEUTZ-JEGHERS SYNDROME IN A CASE OF DIFFUSE HISTOPLASMOSIS*

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RECENTLY a patient was encountered who clinically presented the features of the Peutz-Jeghers syndrome but the intestinal manifestations proved to be inflammatory nonadenomatous pseudopolyps. The rarity of such an occurrence seems to warrant this case report.

REPORT OF A CASE

The patient (Ch.B.) was a sixty-three year old white male who was admitted to the Mt. Alto Veterans Hospital, Washington, D. C., on June 13, 1955. His chief complaints were diarrhea, anorexia and weight loss. The patient stated that since 1918 he had been suffering from diarrhea consisting of four loose stools daily. The stools occasionally had an admixture of a small amount of mucus but never any blood. Throughout these years once monthly or bimonthly he would experience a one day exacerbation of his diarrhea to ten or fifteen stools. About four months prior to admission the number of stools began to increase gradually without any remission, to the point where he was having about ten stools daily. The stools were watery, light in color and contained mucus. The patient experienced rectal urgency and occasionally burning on urination. He did not have any abdominal cramps or discomfort. Nocturnal dyspnea had first been noted about four months previously. A gradual loss of weight amounting to 43 pounds was noted during the four months interval. His appetite, however, remained good until one month prior to admission.

The family history revealed no evidence of gastrointestinal disease in either parent or any of the other three siblings.

The past history included Army Service in World War I and being gassed in 1918. For several years thereafter he had a chronic nonproductive hacking cough which cleared spontaneously. Intermittently, for the past thirty years the patient had suffered from some aching in the knees and elbows. This aching was increased in damp weather and improved with heat. In 1932 both tympanic membranes ruptured, with associated bleeding from the ears for a few days, but without any effect on his hearing. In 1940 he had a "heart attack" which kept him away from work for six months.

Physical examination. The patient was a chronically ill white male who appeared to have lost weight. He weighed 158 pounds. His temperature was 99.4°F., pulse 92, respiration 16, blood pressure 140/70 in the right arm and 110/60 in the left arm. The positive pertinent findings included patchy pigmentation in both upper and lower lips (Fig. 1*A*). Irregular darkly pigmented spots, 1 to 4 mm. in diameter were scattered over the buccal mucosa (Fig. 1*B*). Both tonsils were enlarged; the right one had prominent crypts covered with exudate, the left had a sessile 3 mm. polypoid nodule with a gray surface which bled on scraping. The liver edge was felt 2 to 3 cm. below the right costal margin; the tip of the spleen was palpable. The lower abdomen was slightly tender, but no masses were palpable. The nails were clubbed. Pinpoint pigmented spots were found on the dorsum of the fingers and hands (Fig. 1*C*).

Roentgen findings. The gastrointestinal study disclosed the gastric folds to be somewhat thickened but there was no definite evidence of polyps. The descending duodenum showed at least three radiolucent areas, 6 mm. in diameter, consistent with polypoid formations. No polyps were discernible in the proximal jejunum. The distal jejunum and entire ileum exhibited many circular filling defects up to 1 cm. in diameter (Fig. 2). Barium enema examination of the colon revealed small round radiolucent areas, 3 to 10 mm. in diameter, diffusely

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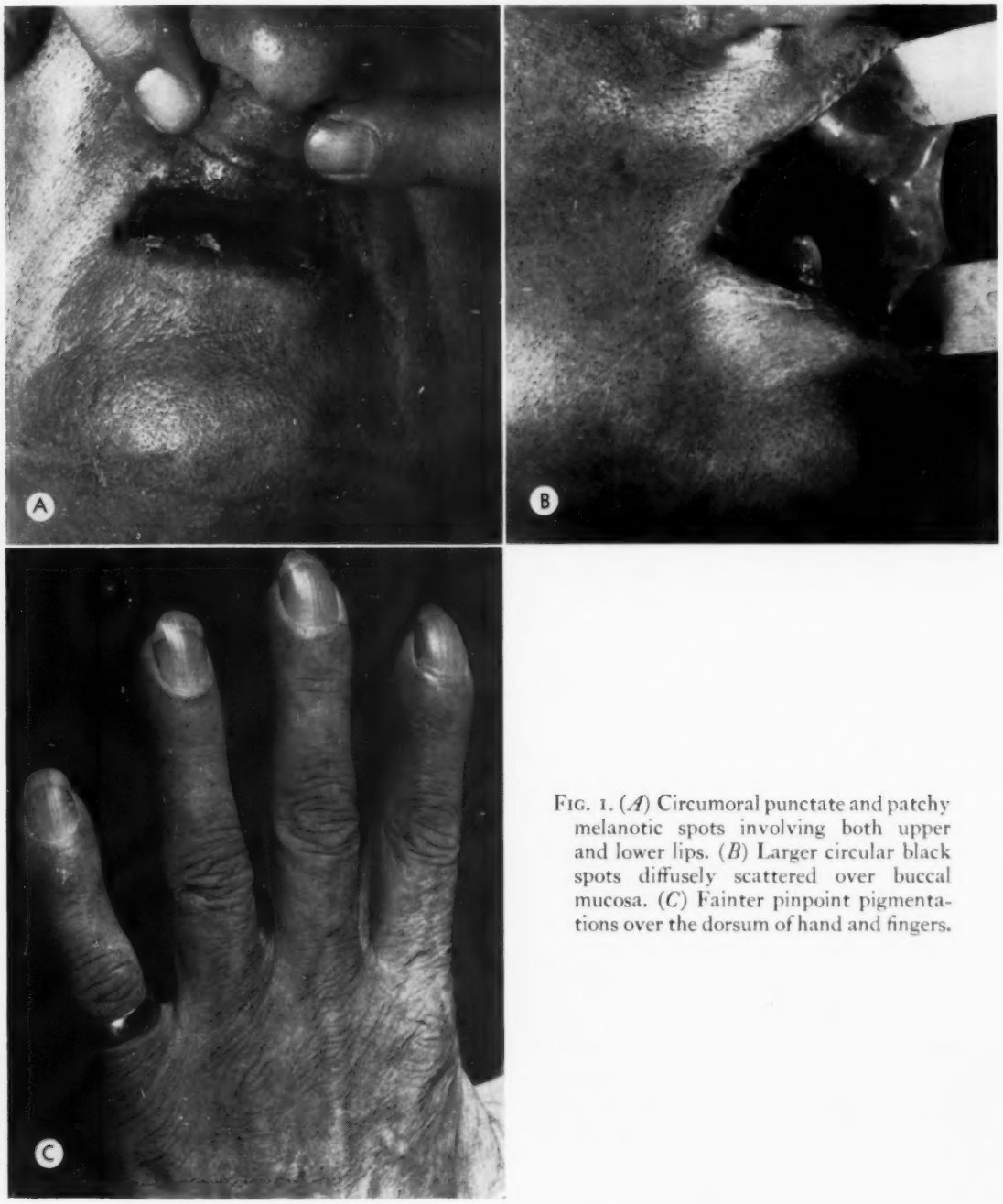

FIG. 1. (*A*) Circumoral punctate and patchy melanotic spots involving both upper and lower lips. (*B*) Larger circular black spots diffusely scattered over buccal mucosa. (*C*) Fainter pinpoint pigmentations over the dorsum of hand and fingers.

involving all segments from the cecum to the sigmoid (Fig. 3). There was no evidence of obstruction, constriction of the lumen or induration of the wall of the colon. The roentgenogram of the chest disclosed opacification of the left apex; a roughly circular 3 cm. density in the left upper axillary region; a semioval 5 by 3 cm. opacity adjacent to the upper left ventricular border; and a 1 cm. density superimposed by the anterior segment of the right fifth rib (Fig. 4). The left pleura was thickened. The heart was normal in size, but moderately retracted to the left with a tented hazy left ventricular margin. Intravenous pyelograms were negative.

Laboratory data. The white blood cell count was 11,000 with 78 per cent polymorphonu-

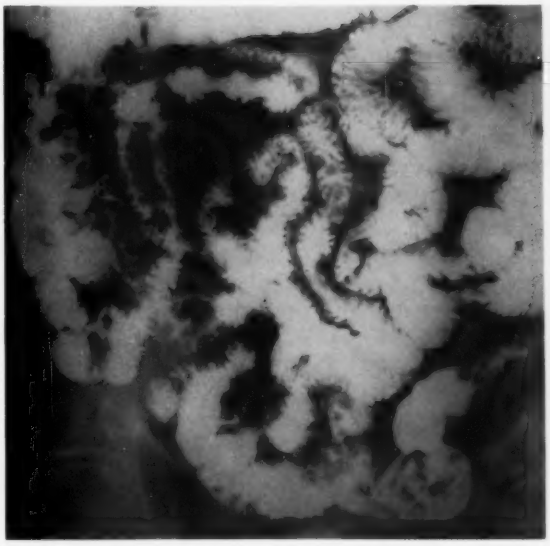

Fig. 2. Small intestinal series showing multiple circular filling defects in the distal jejunum and in the entire ileum.

clears of which 14 per cent were stabs, 17 per cent lymphocytes, 3 per cent monocytes, 1 per cent eosinophils and 1 per cent basophils. Hemoglobin was 16 gm., hematocrit 47 per cent, sedimentation rate 31 mm. per hour. Total eosinophil count was 708 per cu. mm. The glucose tolerance test yielded a diabetic-shaped curve with a peak of 225 mg. per cent in one and one-half hours. Feces were negative for occult blood, ova or parasites. No pathogenic bacteria were isolated on stool or blood culture.

Fig. 4. Roentgenogram of the chest taken on June 16, 1955 revealed patchy opacities in the left apex, upper axillary and paracardiac regions. A small circular density was also present subjacent to the anterior segment of the right fifth rib.

The complement fixation test for Histoplasma capsulatum was positive in a dilution of 1:32.

Gastroscopy. This revealed an abnormal mucosa with an appearance of being infiltrated and presenting a luminous pinkish hue with streaky areas of purulent looking material. No discrete polyps were visualized, nor any ulcerations.

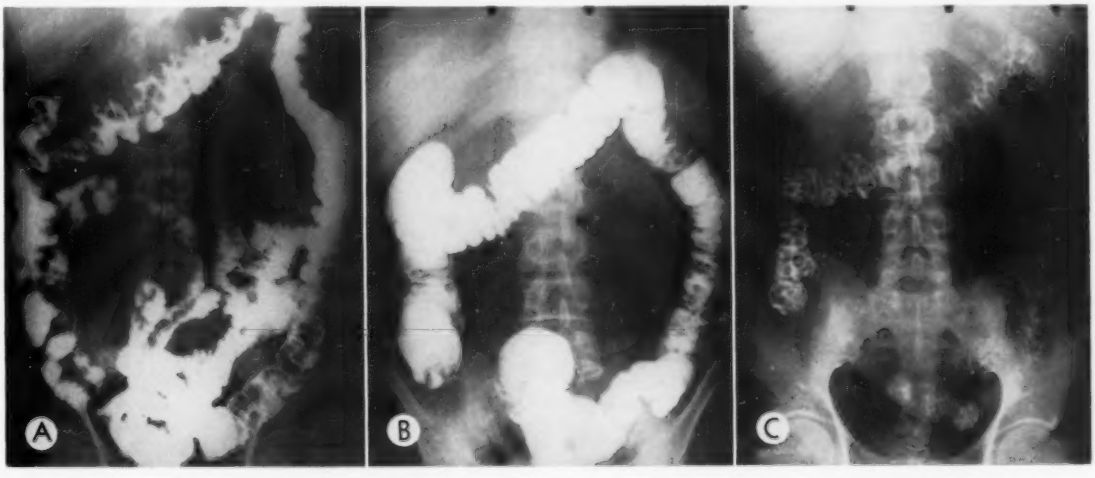

Fig. 3. The circular radiolucent areas diffusely scattered throughout the colon representing polypoid formations were demonstrated: (*A*) On the one and one-half hour gastrointestinal film; (*B*) in the filled phase of the barium enema examination; and (*C*) in the post-evacuation phase of the colon.

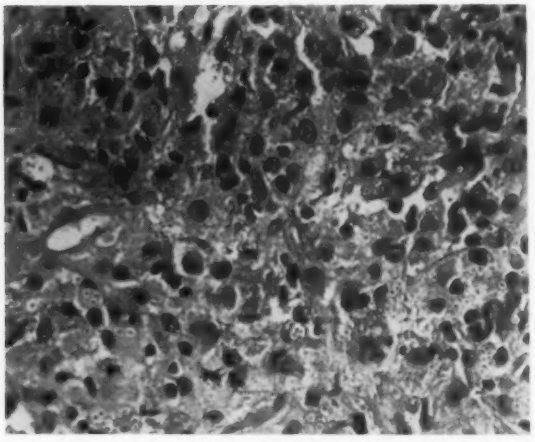

FIG. 5. High power photomicrograph of a segment of a rectal polyp showing numerous organisms typical of Histoplasma capsulatum.

Sigmoidoscopy. This showed numerous polypoid lesions, 2 to 4 mm. in diameter, scattered throughout the rectum, but more numerous in its distal portion. The tops of these polyps were grayish and could readily be scraped off.

Biopsy. Biopsies were taken of the left tonsillar and of the rectal polyps and a needle biopsy was obtained of the liver. These yielded material which was characterized histologically by numerous macrophages filled with organisms typical of Histoplasma capsulatum (Fig. 5). The same organism was also isolated in cultures of bronchial washings.

Hospital course. This was steadily downhill. On July 1, 1955 the patient was transferred to the Clinical Center of the National Institutes of Health, Bethesda, Maryland, for a trial of new drugs. He did not respond to any therapy and died on Oct. 14, 1955.

Autopsy. There was evidence of widespread histoplasmosis with involvement of the tongue, faucial tonsils, pharynx, larynx, trachea, thyroid gland, a parathyroid gland, esophagus, stomach, small intestine, large intestine, rectum, adrenal glands, kidneys, ureters, prostate gland, seminal vesicles, liver, spleen, lymph nodes, brain, spinal cord and bone marrow. The adrenal glands showed extensive destruction, which was in accord with the clinical findings of terminal adrenal insufficiency.

The lesions of the small and large intestines were of special interest in view of the roentgenologic findings of multiple polyposis. Large numbers of polypoid structures measuring 1 cm. in average diameter (Fig. 6) with ulcerated mucosal surfaces and short stalks were found in the large and small intestines. Low-power photomicrographs of two of these polypoid lesions are shown in Figure 7. Microscopically, the mucosa was necrotic and ulcerated and large numbers of macrophages as well as plasma cells and lymphocytes were present in both submucosa and mucosa. Enormous numbers of Histoplasma capsulatum could be seen within the macrophages in the hematoxylin and eosin stains and these were demonstrated more clearly in sections prepared with the periodic acid Schiff and the Gomori methenamine silver reactions. Thus, the colonic polypoid lesions were interpreted as inflammatory pseudopolyps. Ulcerated pseudopolyps were also found in both ureters.

DISCUSSION

In a recent article Cronkhite and Canada[2] concluded: "On the basis of present concepts, it is impossible to classify gastrointestinal polyposis in more than a superficial fashion: familial (polypoid adenomatosis of the colon and Peutz-Jeghers syndrome) and nonfamilial (single adenomatous polyps, multiple adenomatous polyps, and generalized gastrointestinal polyposis)." The difficulty lies in the occurrence of the transition forms and "formes frustes" lacking one or more of the features in the definition of the syndrome. Polyps in most of the hollow abdominal viscera have been reported in association with various pigmentary disorders. Peutz[6] in his original report included nasopharyn-

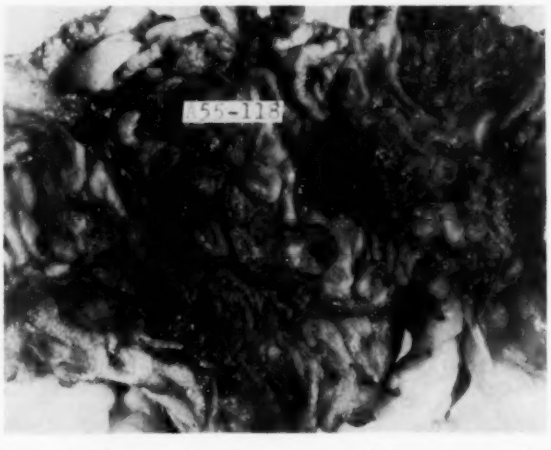

FIG. 6. Photograph of a segment of the gross specimen of the colon depicting the numerous polypoid structures, some with ulcerated mucosal surfaces.

geal polyposis. However, in current usage, the Peutz-Jeghers[5] syndrome consists of circumoral labial, intraoral mucosal and digital pigmented melanotic spots associated with polyps of the small intestine. The concomitant features are: episodes of obstruction, intussusception, rectal bleeding and tendency to malignant degeneration. In about 70 per cent a familial occurrence of this syndrome was established.

In 1953 Gardner and Richards[3] delineated a related familial syndrome consisting of multiple polyposis of the colon, osteomatosis and soft-tissue tumors. An additional related but nonfamilial syndrome was reported in June, 1955 by Cronkhite and Canada.[2] This syndrome included gastrointestinal polyposis, pigmentation, alopecia and onychotrophia.

Though clinically our patient had many of the features of the Peutz-Jeghers syndrome, histologically the intestinal lesions proved to be nonadenomatous inflammatory pseudopolyps. Pseudopolyposis of the gastrointestinal tract is found in: (a) Ulcerative colitis, of specific and nonspecific etiology, the most common variety; (b) regional enteritis; (c) inflammatory fibroid polyps of the stomach and colon. In 1953 Helwig and Ranier[4] reviewed such granulomatous polyps of unknown etiology found in the stomach. In June, 1955 Vitolo and Rachlin[8] reported the first case of a large solitary inflammatory fibroid polyp of the colon; and (d) pseudopolyps of parasitic etiology. Bensaude *et. al.*[1] illustrated a case of bilharzia polyposis. These authors stated that intestinal polyposis caused by bilharzia was found in Egypt and the Antilles, and that in France such observations were reported by Letulle and by Courtois-Suffit. In the same article they also mentioned, without illustrations of histologic proof, their case of rectosigmoid polyposis on the basis of amebiasis.

To the above varieties we are adding our case of pseudopolyposis of the small and large intestines caused by histoplasmosis. Shull[7] in 1953 reported 1 case of diffuse histoplasmosis in which on sigmoidoscopy and at autopsy numerous polypoid nodules were found in the ileum and colon. However, these polypoid granulomata were not demonstrated roentgenologically. The history of colitis with episodes of diarrhea of many years' duration in our patient suggested the diagnosis of Peutz-Jeghers syndrome with superimposed histoplasmosis. However, the histologic findings of nonadenomatous inflammatory pseudopolyps negated such an assumption. We are, therefore, confronted with a patient presenting clinical and roentgenologic features of the Peutz-Jeghers syndrome predicated upon or in association with diffuse histoplasmosis. The interference with intestinal absorption which undoubtedly existed in our patient and the diffuse destruction of the adrenals by the histoplasmosis lesions, raise

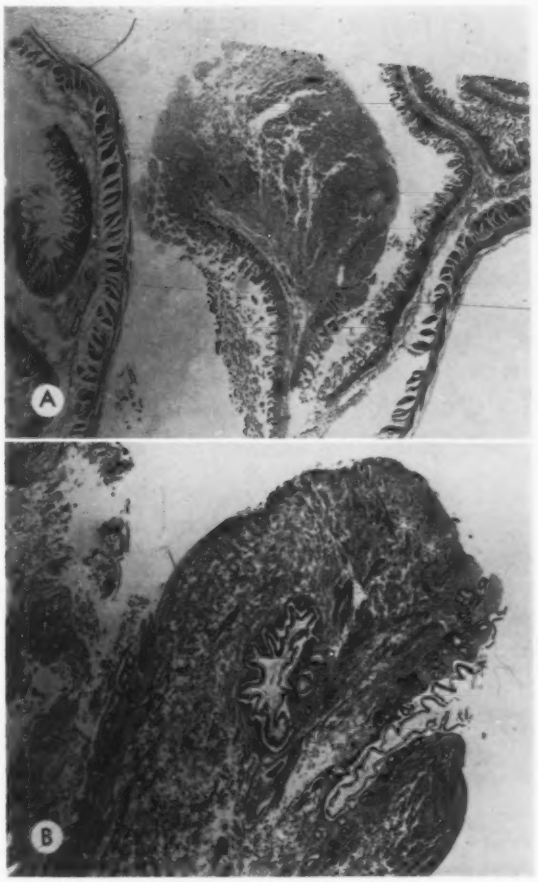

Fig. 7. Low power photomicrographs of two polypoid structures demonstrating that they are not adenomatous polyps but rather inflammatory pseudopolyps. (*A*) Jejunum; (*B*) colon.

the problem of whether these factors were pathogenetically related to the concomitant pigmentary aberration. Unfortunately it was impossible to determine when the circumoral and intraoral mucosal pigmented spots made their appearance. Nevertheless, it seems quite possible that our case represents one in which histoplasmosis has resulted in inflammatory pseudopolyposis of the intestines plus terminal Addison's disease with pigmentations giving a clinical picture of the Peutz-Jeghers syndrome.

SUMMARY

1. A case is reported which clinically presented the Peutz-Jeghers syndrome but in which histologically the intestinal lesions proved to be nonadenomatous inflammatory pseudopolyps due to disseminated histoplasmosis.

2. The question is raised whether the interference with intestinal absorption and the destruction of the adrenals by histoplasmosis might be pathogenetically related to the pigmentary aberration.

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ORAL RENOGRAFIN: A NEW CONTRAST MEDIUM FOR GASTROINTESTINAL EXAMINATIONS*

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THE continuing search for an improved medium in the roentgenologic examination of the gastrointestinal tract has led to the use of a new radiopaque material. This report gives our experiences with oral renografin solution 76 per cent (Squibb) in the examination of the upper and lower gastrointestinal tract. Bismuth was the original medium employed for this purpose but it was soon replaced by barium sulfate which was more satisfactory. In the past few years there have been reports concerning the advantage of smaller and smaller particles of barium.[1,18] Other workers feel that there is no benefit from using barium with a particle size less than that of barium sulfate, USP.[5,16]

Davis, Huang and Pirkey[4] and Shufflebarger *et al.*[16] have defined the requirements of an ideal gastrointestinal roentgenographic medium which are: It should be nonabsorbable from the gastrointestinal tract; it should be nontoxic if accidentally introduced into the body cavities; it should be easy to administer; and it should insure adequate visualization of the mucosa.

The literature contains many reports of accidents due to the administration of barium sulfate as well as of its other disadvantages. Among such actual and potential hazards that have been reported are the accidental introduction of barium into the peritoneal cavity through a ruptured ulcer during an upper gastrointestinal examination,[10] the perforation of the colon during enema examination, and pulmonary embolism.[11,15] The danger of aspiration of barium in examinations of children with tracheo-esophageal lesions and pyloric stenosis is well known. Occurrence of barium granuloma in the rectum[17] and the appendix[13] is also well documented.

Crandall[3] in a series of experiments with rats indicates that barium is not as innocuous as currently believed. He suggests that enough barium is taken into the blood stream by absorption to affect the motility of the bowel. He goes further in his report and suggests the use of zirconium dioxide as a medium for roentgenographic examination of the gastrointestinal tract.

In the study of small bowel obstruction, it has been proved advantageous to employ oral barium, or barium instilled through a tube.[9] In these cases, the use of a medium which would not increase the obstruction and at the same time would lead to an adequate diagnosis without hindering the surgical procedure would be of definite advantage. Epstein[7] has recently described the use of oral iodinated solutions in this regard. Canada[2] has reported the use of urokon (Mallinckrodt) and others[4] the use of hypaque (Winthrop) in the roentgen study of the gastrointestinal tract. In employing these substances, it has been noted that the density is less than that of barium sulfate and that the surface coating of the mucosa is less adequate. The extreme bitterness of these substances has made the insertion of a tube obligatory when administered by mouth. The absorption of urokon 70 per cent and its excretion by the kidneys is another distinct disadvantage of its use.

Oral renografin solution 76 per cent (Squibb) is an aqueous solution of the soluble salt of 3,5-diacetylamino-2,4,6-triiodobenzoic acid. For oral administration it is lemon flavored. It is identical with renografin 76 per cent (Squibb) marketed for intravenous use. The pharmacology and animal toxicity studies have been reported by the Squibb Institute for Medical Re-

* Oral Renografin 76 per cent solution furnished for this study by The Squibb Institute for Medical Research (E.R. Squibb & Sons, New Brunswick, New Jersey).

search and this report is available upon request. The use in man has been reported by Lentino *et al.*,[12] Eyler *et al.*,[8] Dennis[6] and Orr and co-workers[14] who employed renografin as a urographic medium. The use of the same medium when given orally would be expected to present little hazard from toxicity.

MATERIAL AND RESULTS OF EXAMINATION

Our series includes 41 routine examinations of the upper gastrointestinal tract (including 1 small bowel study and 3 esophageal studies) and 6 colon examinations (one hesitates to use the term "barium enema"). The medium was used undiluted in 23 cases and was diluted 1:1 in 18 cases in which the upper gastrointestinal tract was studied. Not over 100 cc. of the total volume was needed for a complete study of the stomach. For the colon examinations, a mixture of 1:1 totaling 600 cc. was used in 3 of the cases. In the other 3 cases the dilution was two parts medium to four parts water. In all except one colon study it was found that 600 cc. was sufficient to fill the entire colon. In 14 of the upper gastrointestinal examinations, previous barium sulfate roentgenograms were available which could be compared with the oral renografin series. In this group, when comparable films were placed side by side, it was difficult to tell which medium had been employed provided the undiluted oral renografin was used. In the cases in which the contrast material was diluted, the roentgenograms were satisfactory, although the density was less than that of the conventional barium sulfate roentgenograms.

In those cases in which renografin was the only medium used, its value was as good as, or superior to, that of barium sulfate in that excellent visualization of the mucosal pattern of the stomach and duodenum was noted at fluoroscopy. In the colon examinations, too rapid filling of the colon was seen but this could be controlled by regulating the gravitational flow of the medium. The initial spasm so often seen in the rectosigmoidal area when using barium sulfate was absent. Also, the ballooning of the rectal ampulla before filling of the sigmoid was not encountered. When using renografin and a high kilovoltage technique, the resulting roentgenograms were superior to those obtained after barium sulfate administration. One of the disadvantages noted in the colon studies was the patient's inability to evacuate the medium too well. It is possible that the loss of a bulk stimulus was responsible for this and that the addition of tannic acid to the medium might aid in overcoming this disadvantage.

The examination of the upper gastrointestinal tract was as satisfactory as with barium sulfate in all cases. However, in the lower small bowel the main disadvantage of the use of renografin was due to its osmotic activity. Because of this, there was dilution of the solution, although the roentgen visibility of the small bowel was not affected. Another disadvantage noted was that in examining the esophagus in the upright position, the medium moved too rapidly and an inadequate visualization resulted. To offset this, oral renografin was combined with a gelatin preparation and this thicker preparation enhanced the visualization of the esophagus. In 20 per cent of the upper gastrointestinal examinations there was noted a reflex spasm at the pylorus after the passage of the medium into the small bowel. This presented no real problem as that region had already been checked by fluoroscopy and spot roentgenograms. Only in one of our cases was there a suggestion of medium in the kidneys.

In the examinations of the colon, no toxicity to the medium was noted. In the upper gastrointestinal series, 2 of the patients complained of significant diarrhea and one patient vomited the contrast material one hour after it was administered. In these cases, the distress subsided without any particular treatment. None of the signs of iodine sensitivity were observed in any of the cases. We feel that the diarrhea is a

result of the osmotic changes in the intestinal tract rather than of primary sensitivity to the iodine medium. All of the patients tolerated the opaque material when given orally and although some complained of the bitterness, several of the patients who had had both media preferred the oral renografin solution to the barium sulfate. There were no complaints of constipation which are so often encountered when using barium sulfate.

CONCLUSIONS

Oral renografin solution 76 per cent (Squibb) is a safe contrast medium which can be used in the roentgenologic examination of the gastrointestinal tract. In many ways its value is superior to that of barium sulfate; namely, in mucosal contrast, tolerance and safety of administration. It presents certain disadvantages in the study of the esophagus and the colon, but these disadvantages can probably be obviated by additional study and research. The problem of diarrhea is present when given orally; however, this problem is much less than that of constipation when barium sulfate is used. We shall continue to investigate the many possibilities that this medium offers in the study of the gastrointestinal tract.

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ORAL RENOGRAFIN 76 PER CENT: A CONTRAST MEDIUM FOR EXAMINATION OF THE GASTROINTESTINAL TRACT*

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ALTHOUGH barium sulfate remains the standard contrast medium for roentgenographic examination of the gastrointestinal tract, the search has continued for an ideal medium, particularly in emergency examinations. Davis, Huang and Pirkey[1] have described the use of urokon in 1956. Recently, oral renografin solution 76 per cent (Squibb) became available and our experiences with this medium serve as a basis for the present report. This substance has been evaluated and compared with barium sulfate, as to its advantages and disadvantages. The indications and contraindications for the use of oral renografin are presented.

PROPERTIES

Oral renografin 76 per cent is a lemon flavored aqueous solution of the solubilized salt of 3,5-diacetylamino-2,4,6-triiodobenzoic acid. This acid, from which the solubilized salt is derived, has a molecular weight of 613.9 with 62.02 per cent iodine. Oral renografin 76 per cent contains 59.7 per cent renografic acid and 37.0 per cent iodine. The solution has a specific gravity of 1.42 at 20° C. and a pH of 6.0–6.5. This solution has a viscosity at 25°C. of 10.4 centipoises. The contrast medium has been tested on dogs with no evidence of toxic effects. Only traces of iodine excretion were recovered in serial urine samples.

CLINICAL MATERIAL

Roentgen examination of different portions of the gastrointestinal tract was carried out on 45 different patients. Undiluted oral renografin 76 per cent was used in most of the cases. A 50 per cent dilution with water produced an adequate radiopacity. No toxic effects were observed. The types of examination and indications for examination are listed in Table 1.

TABLE I

TYPES OF CASES IN WHICH ORAL RENOGRAFIN 76 PER CENT HAS BEEN USED

Megacolon	3
Large bowel obstruction	15
Upper gastrointestinal obstruction	18
Postoperative cases—resection or perforation of gastrointestinal tract	9
Gastrointestinal bleeding	6
Foreign body	2

Figures 1 through 12 illustrate the various kinds of cases in which this medium has been effectively used. Two very interesting findings were observed in this series of cases. In 2 instances there was a significant excretion of contrast medium through the urinary tract, permitting excellent visualization of the kidneys and urinary bladder. The other finding was a marked dilution of the contrast medium by small intestinal fluid which rapidly diminished the radiopacity of the contrast medium, militating against adequate mucosal study in the small intestine.

DISCUSSION

The properties for an ideal contrast medium for use in roentgenographic examination of the gastrointestinal tract have

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Fig. 1. Carcinoma of the mid-descending colon demonstrated by renografin 76 per cent, administered via rectum as an enema in an emergency examination.

Fig. 3. Pyloric stenosis in infant with area of organic narrowing demonstrated after oral administration of renografin 76 per cent. Not as clearly outlined as with barium. Note dilution effect in small bowel.

Fig. 2. Megacolon with site of aganglionosis clearly seen during rectal instillation of renografin 76 per cent. Large scybalum demonstrated in pelvic colon with area of spasm directly above.

been recorded by Davis, Huang and Pirkey.[1] Such an opaque medium should have the following attributes: 1. The medium should remain in solution without settling out and permit a uniform radiopacity; 2. the medium should preferably be water soluble but nonabsorbable from the intestinal tract; 3. the medium should not have the potential of becoming inspissated in the intestinal tract (as barium sulfate); and 4. the medium should be nontoxic, if absorbed.

Although other water soluble contrast media have been used in examination of the gastrointestinal tract, oral renografin 76 per cent is the first such opaque medium to

FIG. 4. Outline of the trachea and bronchial tree by renografin 76 per cent given orally in case of laryngeal carcinoma. A water soluble contrast medium is obviously safer than barium sulfate in such instances.

be prepared specifically for this use. This medium is more palatable, permitting direct ingestion rather than necessitating instillation through a tube into the stomach or small intestine.

FIG. 6. Opacification of stomach, duodenum and rest of small intestine with orally administered renografin 76 per cent. The dilution effect in the small intestine is clearly demonstrated.

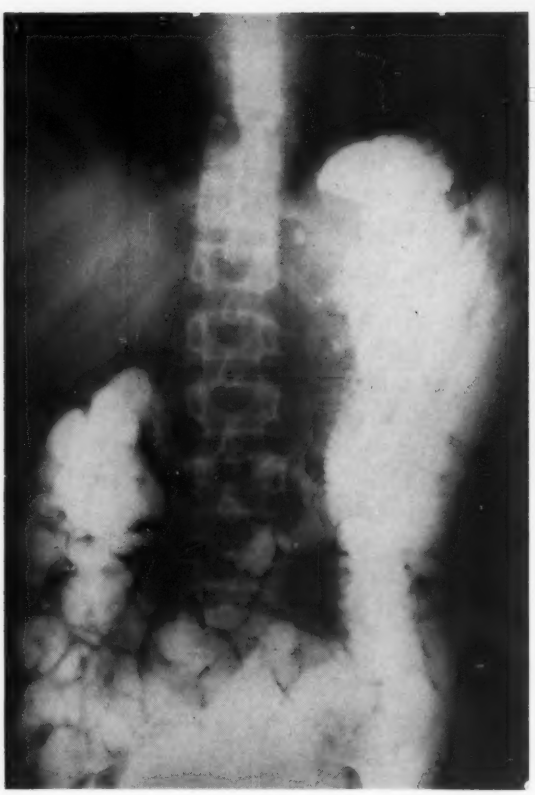

FIG. 5. Outline of stomach residual and small intestine after partial gastrectomy for ulcer. The "smudgy" effect in the small bowel is due to excessive dilution. Note the faint opacification of the collecting system of the right kidney.

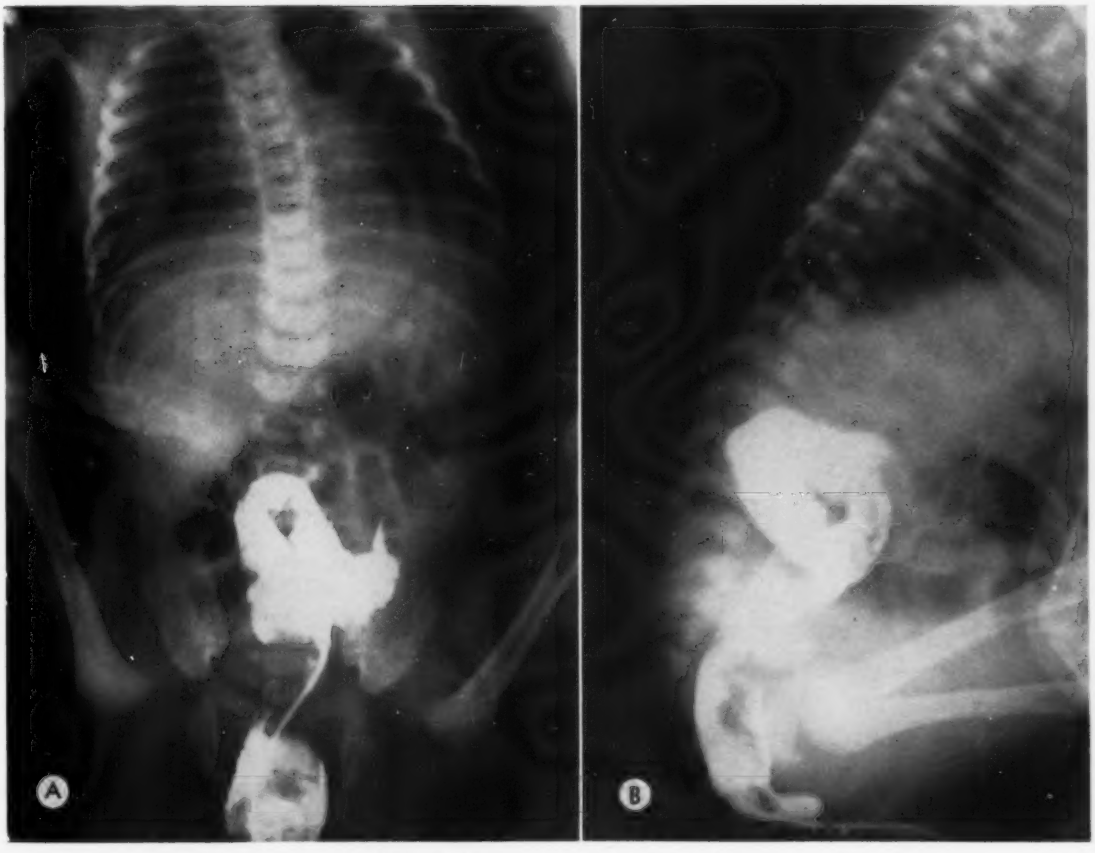

Fig. 7. (*A*) Rectovaginal fistula demonstrated via renografin enema—frontal plane. (*B*) Same case. Lateral view.

Advantages. Oral renografin 76 per cent is water soluble and of low viscosity with satisfactory radiopacity. No toxic effects were noted. Previous studies have already demonstrated the safety of renografin as a contrast medium in intravenous urography.[2,3] The solution is uniform in its radiopacity with no settling on standing. It is readily available and requires no preparation.

Disadvantages. The dilution of renografin 76 per cent in the small intestine does not permit a satisfactory visualization of the mucosa of the small bowel. The demonstration of absorption of the contrast medium via the urinary tract raises the possibility of systemic toxicity. The relatively low viscosity of the medium might permit its passage through zones of incomplete obstruction more readily than barium. This may be an advantage or disadvantage. Renografin 76 per cent is, at present, considerably more expensive than barium.

Indications. The indications for the use of renografin 76 per cent are:

1. Acute bleeding from the upper or lower gastrointestinal tract.

2. Obstruction of the colon with examination via the rectum as an emergency procedure to localize and/or confirm the site of obstruction.

3. Obstruction of the stomach or small intestine. If the renografin passes readily into the large bowel, then a follow-up examination is permissible with barium in order to exclude any partially obstructing lesion.

4. Postoperative intestinal resection cases (immediate) where the use of barium

FIG. 8. Opacification of collecting system of both kidneys by renografin, showing marked left-sided pyelocaliectasis, probably secondary to inflammatory process in pelvis. This roentgenogram was exposed ten minutes after the patient evacuated renografin 76 per cent which was administered as an enema to outline the area of diverticulitis. Some areas of small intestine (reflux) remain opacified with renografin.

might be dangerous in terms of its weight and potential of leakage.

5. Cases of impending perforation of the gastrointestinal tract, *i.e.*, peptic ulcer, diverticulitis, etc., where the use of barium might be potentially hazardous.

6. Foreign bodies in the gastrointestinal tract, particularly in the pharynx and esophagus. When renografin 76 per cent is used, the foreign body is not obscured for subsequent endoscopy, as may occur with the use of barium, and the pharyngeal or esophageal walls are not coated, as with barium.

FIG. 9. Carcinoma of sigmoid colon outlined during renografin enema. Renografin was used because the patient was clinically obstructed.

FIG. 10. Duodenal bulbar ulcer (faintly outlined) diagnosed in patient with massive hematemesis with orally administered renografin.

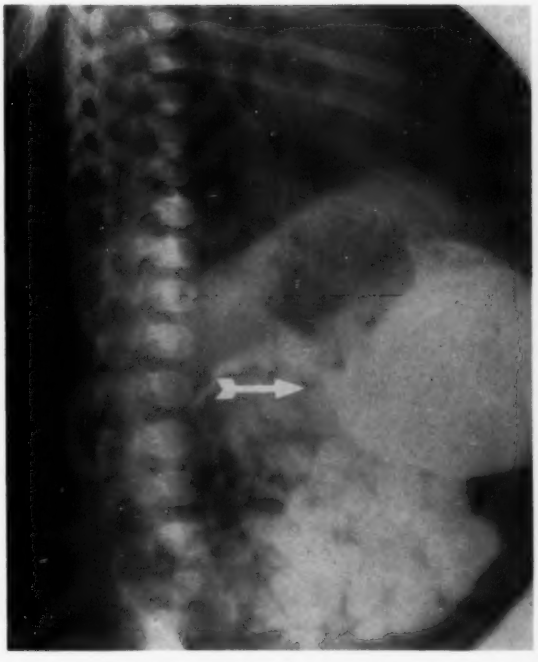

Fig. 11. Pyloric stenosis diagnosed in infant with use of oral renografin.

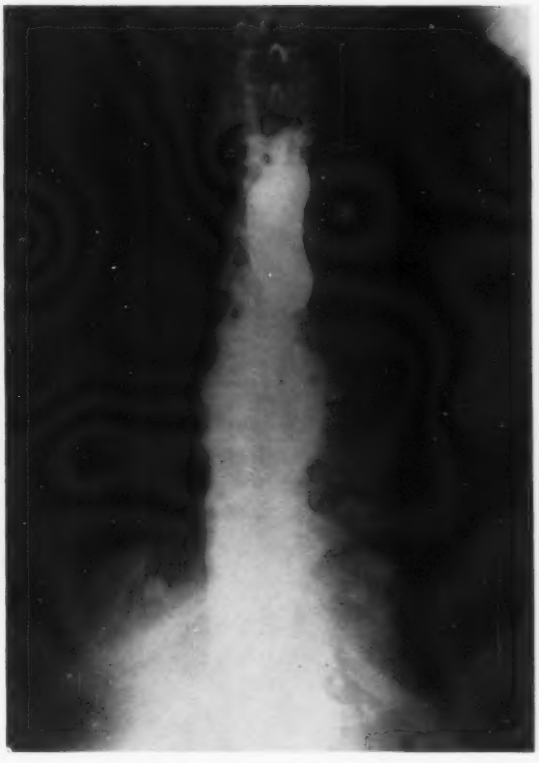

Fig. 12. A typical carcinoma of the mid-third of the esophagus, delineated with oral renografin in a patient with symptoms of complete obstruction. Aspiration of renografin in such a case would be less hazardous than with barium.

7. Sinus tracts and fistulae of undetermined course, where barium might be potentially dangerous.

8. Abscess cavities.

9. Postoperative examination of the intestinal tract to differentiate mechanical from inhibition ileus.

10. Cases of megacolon, where stasis of contrast medium such as barium may result in damaging inspissation. The diminished viscosity of the oral renografin 76 per cent permits more ready evacuation or siphoning of the contrast medium. This contrast medium should reduce the possibility of shock associated with excessive water retention.

11. Examination of the upper gastrointestinal tract in infants, where the possibility of stenosis, atresia or fistula exists. In such instances, the potential danger of aspiration of contrast medium into the lung is always present. Oral renografin is then obviously preferable to barium sulfate.

12. Cases of disordered swallowing mechanism, *i.e.*, central nervous system abnormalities, neuromuscular disturbances, obstructing lesions of the pharynx and esophagus, etc.

Contraindications. We are not aware of any significant contraindications to the use of oral renografin 76 per cent in the gastrointestinal tract. Because of the excessive absorption seen in 2 cases, one might raise the question of using this medium in instances of known severe hepatocellular and renal cell damage.

Limitations. These are:

1. The dilution effect that occurs in the small bowel with oral renografin 76 per cent militates against the use of this medium in demonstrating any but the grossest lesions in the small intestine.

2. The diminished viscosity of this medium has served as a disadvantage in demonstrating pyloric stenosis in infants. We have not reached a definite conclusion

in determining the effect of this diminished viscosity in the demonstration of peptic ulcers.

3. The cost of this medium, particularly in an undiluted form, is certainly considerably greater than that of barium sulfate. As further experience is gained, this cost may be reduced by further dilution.

CONCLUSION

Oral renografin 76 per cent is a satisfactory opaque medium for roentgenographic examination of the gastrointestinal tract. It has been used in a number of conditions requiring an emergency study. The safety of this contrast medium has been established. The advantages, disadvantages, indications, contraindications and limitations of its use have been discussed.

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DIOCTYL SODIUM SULFOSUCCINATE (DOXINATE*) AS AN ADJUNCT IN THE ROENTGENOLOGIC INVESTIGATION OF THE GASTROINTESTINAL TRACT—A CLINICAL STUDY

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DEHYDRATION of barium sulfate suspension in the large intestine, causing discomfort, impaction and even obstruction, has long been a major difficulty following roentgenologic examination of the gastrointestinal tract. To overcome this, patients were advised for many years to take laxatives, mineral oil preparations and/or enemas. These measures were not universally accepted for various reasons. The availability of a fecal softening agent, dioctyl sodium sulfosuccinate (doxinate—hereafter referred to as DSS) led us to investigate its effectiveness in maintaining softness and homogeneity of the barium fecal mass and inducing elimination without discomfort or impaction. The results have been highly gratifying and we now use this preparation as a part of our routine in gastrointestinal examinations by ingested barium sulfate-water suspension.

Dioctyl sodium sulfosuccinate (DSS) is a material which reduces surface tension between aqueous and solid or semisolid constituents of the bowel, thus forming a more homogeneous mixture, preventing dehydration and increasing bulk of the fecal mass. It has been used successfully in the treatment of chronic and acute constipation in geriatric,[4,6] bed-ridden,[10] pediatric,[9,10] postpartum,[5,7] immobilized,[10] and other types of patients.[1,3,4,6,8] It is nontoxic, noncathartic[2,10] and produces a soft stool which is evacuated easily.

Our interest in this preparation began in June of 1956. Since then we have employed it in various forms in the examination of more than 750 patients in a private office practice and 50 patients in the Out Patient Department of the United States Veterans Administration Hospital, Regional Office. Because of the nature of patient material, a complete follow-up study has not been possible, but we have obtained information from a representative number of cases by verbal questioning, and some patients cooperated by returning for twenty-four, forty-eight, and, in a few instances, seventy-two hour checkup examinations.

The initial investigation was with the use of 60 mg. soft gelatin capsules of DSS. For many years we have pursued a routine of administering a so-called "motor meal" three hours prior to the regular examination. Patients were given an envelope containing 1½ ounces of barium sulfate with instructions to stir it up in a glass of water and drink it three hours prior to coming for examination. The emptying time of the stomach was determined by either fluoroscopy or roentgenography, or both. Additional barium-water was given for roentgen study of the stomach and small intestine. Thereupon they were given a 60 mg. capsule of DSS and instructed to obtain six more capsules from the pharmacy and take one each morning until all were used. Two patients complained of difficult and uncomfortable evacuation, and it was presumed that the optimal effect was not obtained because of the "head start" of the initial barium-water suspension, three hours prior to ingestion of DSS.

We then learned of the availability of a 5 per cent liquid DSS preparation, whereupon we tried mixing 5.0 cc. in the barium sulfate "drink." Some of these patients re-

* Clinical supplies of doxinate, 5 per cent solution, 60 mg. and 240 mg. soft gelatin capsules, were obtained through the courtesy of Lloyd Bros., Inc. Cincinnati, Ohio.

ceived a "motor meal" and others came for examination on a fasting stomach. The results in all of these patients were considerably better, presumably because the DSS was dispersed more promptly through the intestinal tract. A few patients were given 10 cc. liquid DSS to determine if the larger quantity would produce still better results; however, this effort remained unsuccessful.

The third group of patients came for examination on a fasting stomach, without having taken the "motor meal" previously. Immediately after completion of the roentgen examination, they were given one 240 mg. soft gelatin capsule of DSS. This has been found to be the most desirable procedure.

RESULTS

Evacuation of the barium sulfate fecal content was facilitated by all of the methods used. The best effect was obtained when the DSS, either in liquid or capsule form, was given with the initial ingestion of opaque material.

DSS in Liquid Form. We found that 5.0 cc. of 5 per cent solution gave more satisfactory results than when 10 cc. was used. Whereas some patients commented that the addition of 5.0 cc. to the unflavored barium sulfate suspension actually improved the taste, those who received the larger quantity invariably complained of the bitterness of the taste. There was no improvement with regard to softness and homogeneity of the fecal mass when the larger quantity was used.

The mixing of DSS in liquid form with the barium-water suspension appeared to us to stimulate peristalsis and, while we did not encounter any cases of "dumping," there were no cases of pylorospasm. We feel that the use of liquid DSS in the opaque suspension might mask a functional pylorospasm. There is also the possibility that the "wetting action" of the DSS might prevent adherence of barium sulfate to the gastric or intestinal mucosa and thus prevent detection of a lesion. These considerations, together with our adherence to the attitude that plain barium sulfate in water is the preferred opaque medium for gastrointestinal roentgenologic investigations, led us to abandon the use of DSS in the mixture.

DSS in Capsules.—The somewhat less favorable results using 60 mg. DSS capsules are probably due to the fact that the preparation was given three hours after the barium sulfate suspension had a "head start." It is conceivable that more satisfactory results would have been obtained if these patients had taken the capsule along with the "motor meal," supplemented by other 60 mg. capsules at twenty-four hour intervals thereafter. This regimen would impose the nuisance, even though slight, of having to remember to take the capsule at a certain time. There is also the consideration of expense, since pharmacies are, generally, unwilling to dispense small quantities of drugs and patients would be left with unused material on hand.

The results with a single 240 mg. capsule of DSS, given immediately following the initial ingestion of the barium sulfate-water suspension, have been universally good. No patients have complained of discomfort, impaction or obstruction and even in case of chronic constipation (Fig. 4) elimination of the barium fecal mass has been comfortable.

As mentioned above, a close follow-up has not been possible because of the private practice type of patient material available. We have been able to secure checkup roentgenograms at twenty-four, at twenty-four and forty-eight, and in a few instances up to seventy-two hours after ingestion of barium and DSS by one or another of the three routines described in approximately 75 cases. In addition, we have questioned a representative number of patients, most of whom had had previous gastrointestinal examinations. All patients questioned preferred the use of DSS, in one form or another, to the older methods.

As an objective illustration, Figures 1, 2, and 3 include reproductions of twenty-four and forty-eight hour roentgenograms from

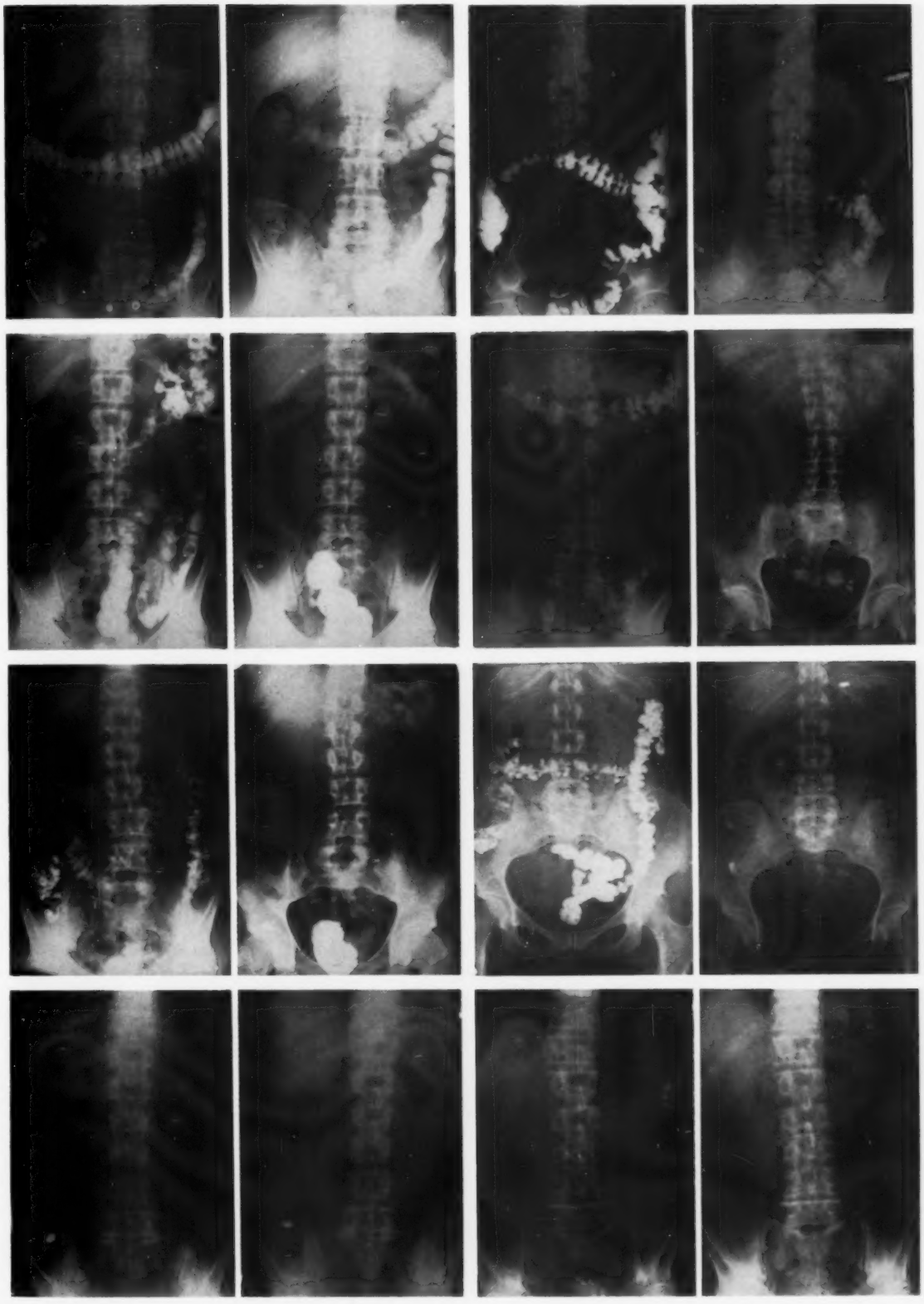

Fig. 1. Eight cases. Twenty-four and forty-eight hour follow-up roentgenograms. Patients were given 5.0 cc. 5 per cent liquid doxinate mixed with the initial barium meal.

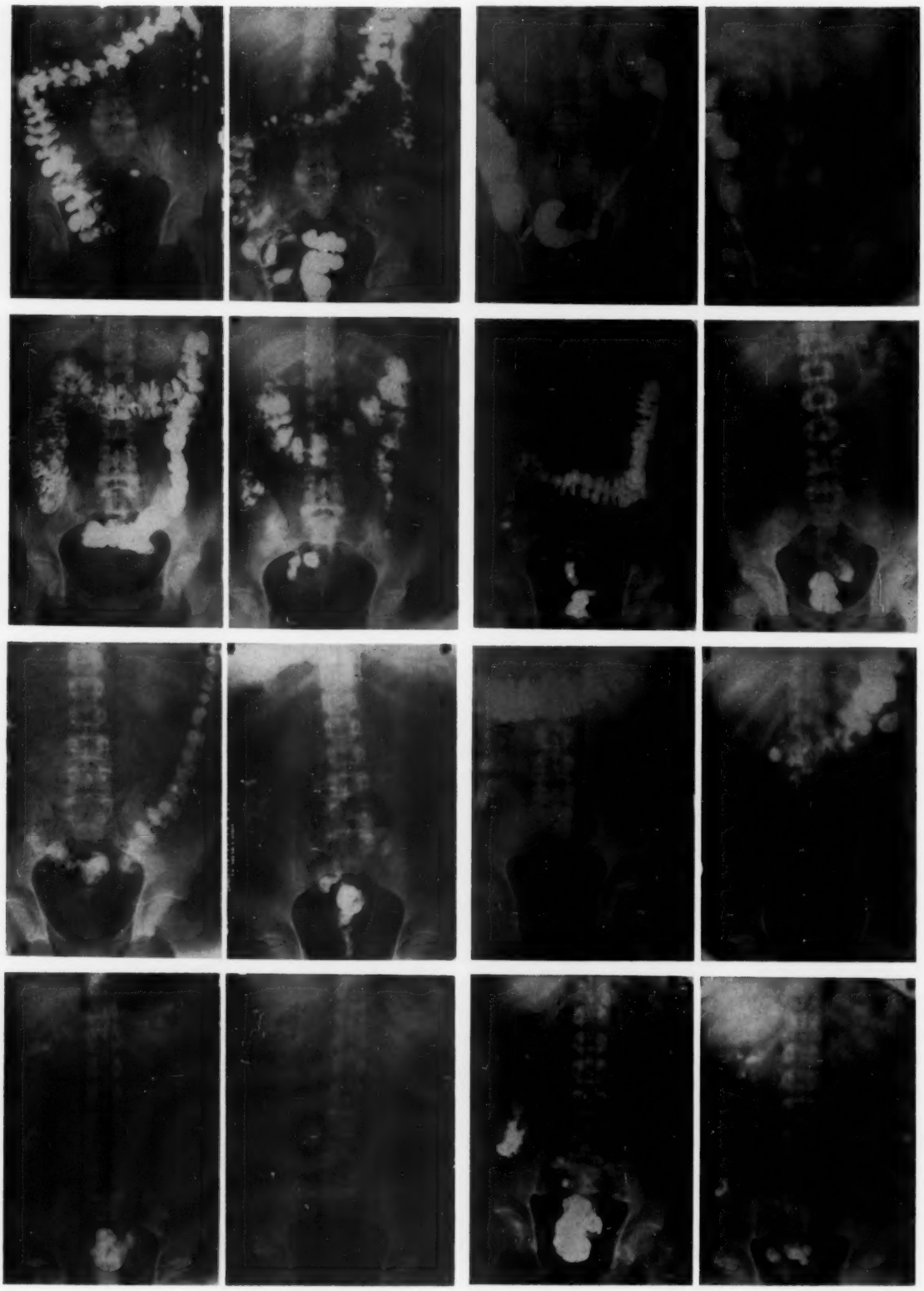

FIG. 2. Eight cases. Twenty-four and forty-eight hour follow-up roentgenograms. Patients were given 240 mg. capsule of doxinate immediately after ingestion of initial barium meal.

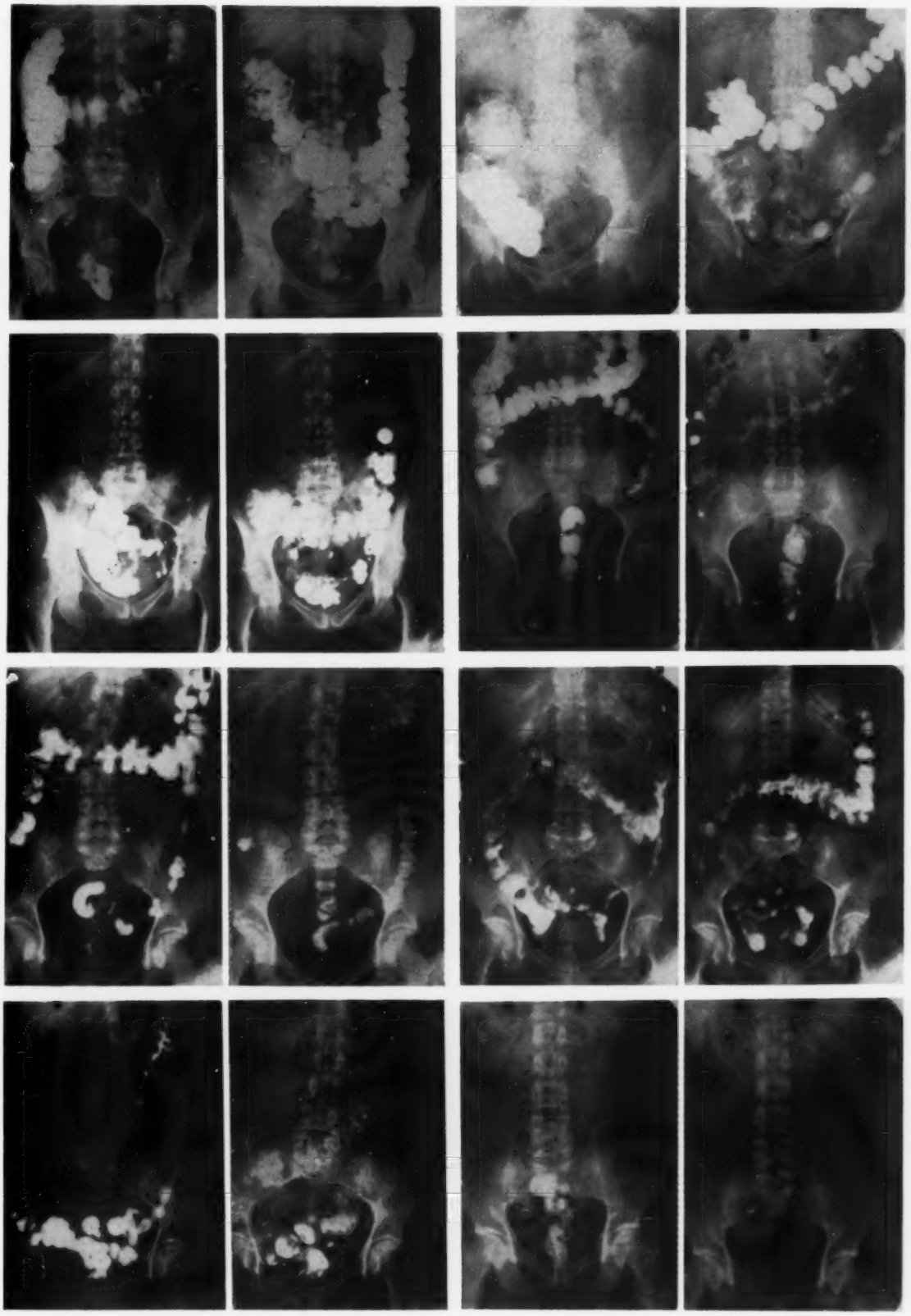

Fig. 3. Eight cases. Twenty-four and forty-eight hour follow-up roentgenograms. Patients did not receive doxinate or other fecal softening medication.

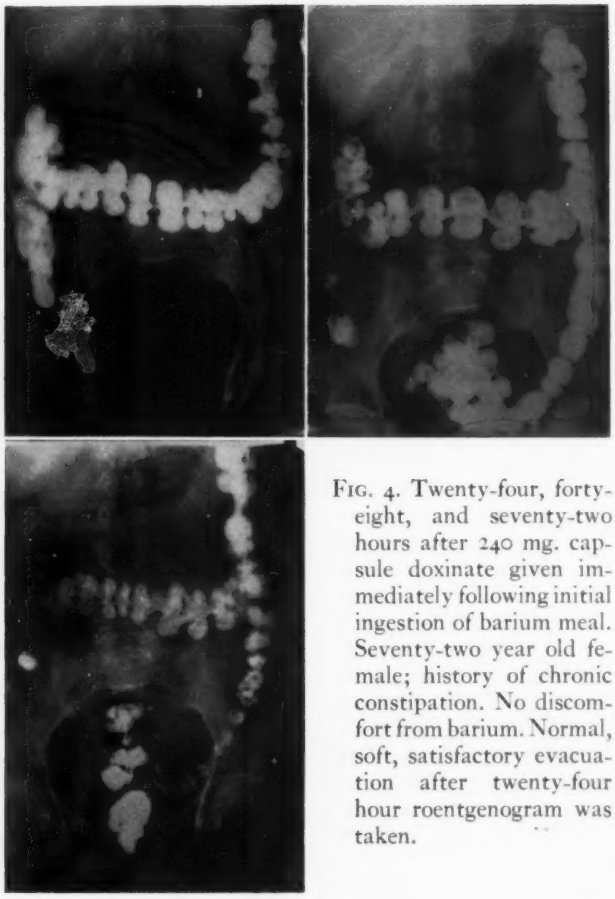

Fig. 4. Twenty-four, forty-eight, and seventy-two hours after 240 mg. capsule doxinate given immediately following initial ingestion of barium meal. Seventy-two year old female; history of chronic constipation. No discomfort from barium. Normal, soft, satisfactory evacuation after twenty-four hour roentgenogram was taken.

an unselected group of patients who were given 5.0 cc. 5 per cent DSS in the barium sulfate-water suspension, a 240 mg. DSS capsule immediately following the initial ingestion of barium sulfate-water, and who received no DSS. It seems to us, from these observations and from others which were made only after twenty-four hours, that the barium fecal mass in the large intestine is softer and of more homogeneous consistency when DSS is used. For this reason, the procedure is advantageous when follow-up studies of the large intestine at twenty-four hours, or longer intervals, are desirable.

SUMMARY

There is evidence that dioctyl sodium sulfosuccinate (DSS) is a useful adjunct in the roentgenologic investigation of the gastrointestinal tract by ingested barium sulfate-water suspension, in that a soft, homogeneous fecal mass is produced. No laxative, toxic or other undesirable side-effects were encountered. Patient acceptance of the drug has been excellent.

The present technique consists of administration of one 240 mg. capsule of DSS at the time of initial ingestion of barium sulfate-water suspension. If the patient takes a "motor meal," a capsule is given to be taken along with it. If the initial barium study is performed in the radiologic department, the capsule is given immediately after the completion of the fluoroscopic and roentgenographic examination.

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A SUGGESTION FOR DIMINISHING THE AMOUNT OF GAS INTRODUCED INTO THE SUBDURAL SPACE DURING LUMBAR PNEUMOENCEPHALOGRAPHY*

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THE presence of gas in the subdural space sometimes results in an unsatisfactory pneumoencephalographic examination. Gas may enter the subdural space through a small rent in the arachnoid caused by a traumatic lumbar puncture. A similar situation may exist if a spinal tap has been done a few days before pneumoencephalography. Another possible mechanism might be such placement of the point of the spinal needle that gas is injected into both the subdural and subarachnoid spaces, outlining the subdural areas as well as the ventricular system.

TECHNIQUE

We have found the following technique helpful in diminishing the handicaps incident to the subdural instillation of gas: The lumbar puncture needle is introduced into the fourth or fifth lumbar interspace. Scout roentgenograms are made and viewed immediately after the instillation of 10 to 20 cc. of gas. If the subtentorial or subdural space is visualized, the needle is withdrawn immediately, and reinserted at least two interspaces higher. When a free flow of cerebrospinal fluid is obtained, the needle is inserted from 3 to 5 mm. deeper, and on continuation of free flow 10 to 20 cc. of gas is instilled. Further scout roentgenograms are then made and the examination continued if air is in the ventricular system.

If a diagnostic lumbar tap had been done within a week preceding lumbar pneumoencephalography, the repeat lumbar puncture is made two interspaces above

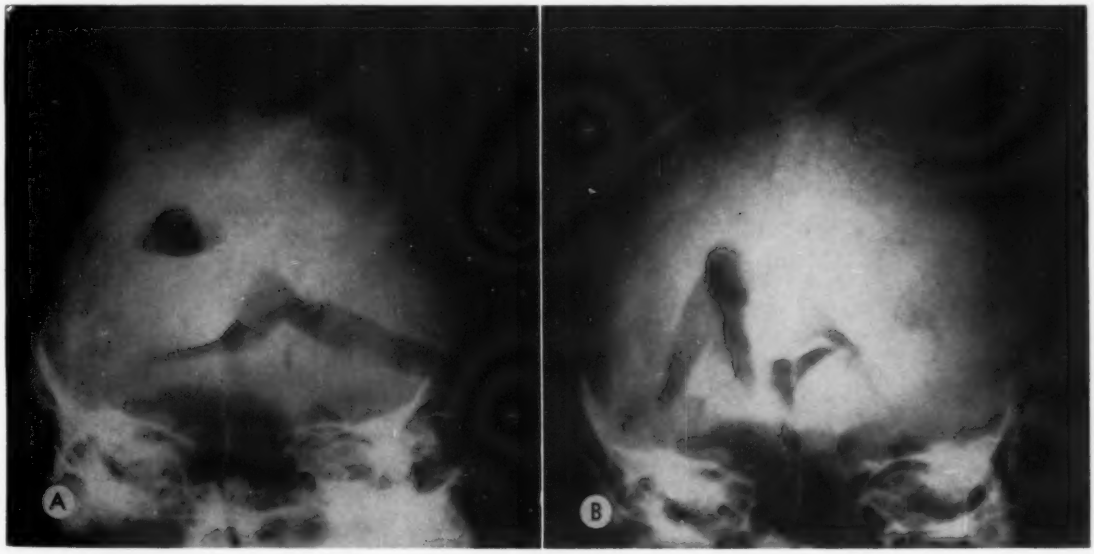

FIG. 1. (*A*) Posteroanterior pneumoencephalogram reveals gas in the subtentorial space. A small amount has entered the right lateral ventricle. (*B*) Same patient after replacement of needle two interspaces higher and lumbar instillation of 30 cc. of atmospheric air. The ventricular system is well filled and discloses a left parasagittal tumor.

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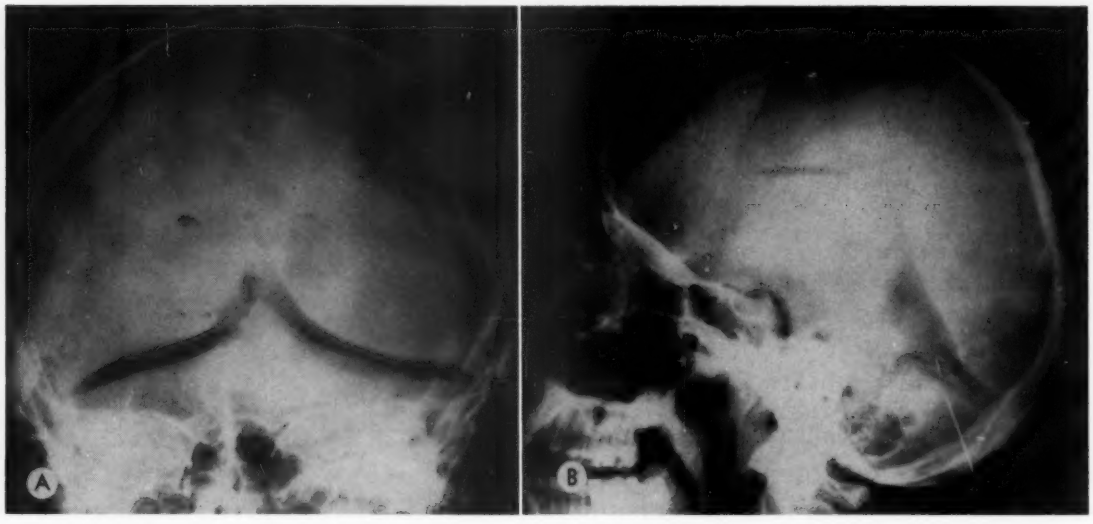

FIG. 2. (*A*) Posteroanterior and (*B*) lateral pneumoencephalograms made after lumbar instillation of 20 cc. of gas. Note the collection of gas under the tentorium, along the floor of the middle and anterior cranial fossae. A small amount of air is present in the ventricles.

the original site. Whenever possible, it is preferable to combine a diagnostic tap with performance of pneumoencephalography to avoid repeating the lumbar puncture. If a myelographic examination becomes necessary, similar care in the placement of the spinal needle should be exercised to avoid introduction of pantopaque into the subdural or epidural spaces.

CASE ILLUSTRATIONS

Two representative cases are illustrated. In the first patient the initial scout roentgenograms showed a considerable amount

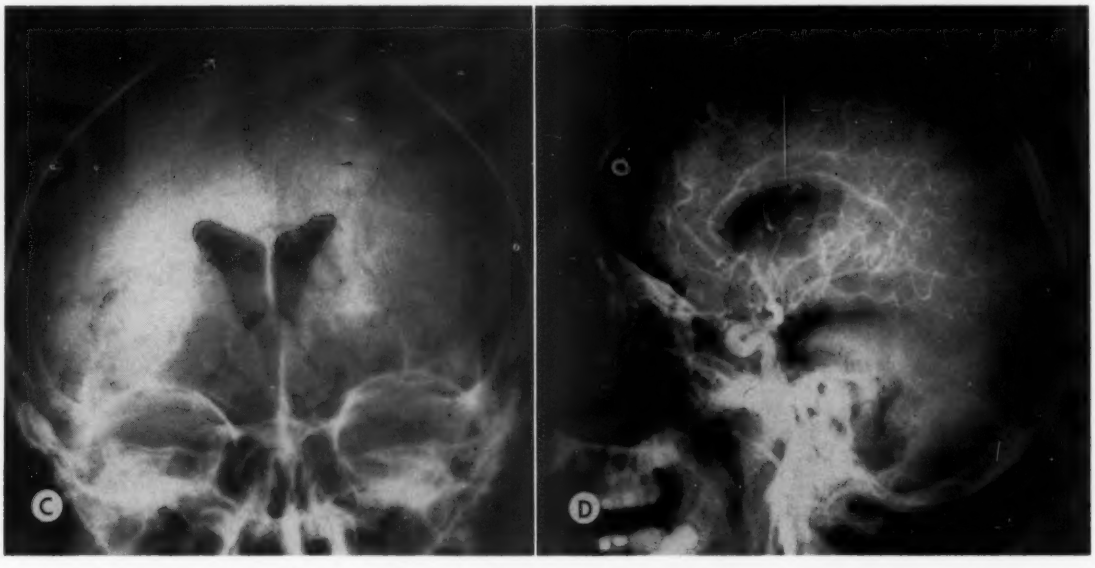

FIG. 2. (*C*) Same patient after removal of the lumbar puncture needle and replacement two interspaces higher. The gas outlines the ventricular system satisfactorily. (*D*) Percutaneous cerebral angiogram in brow-up position. The subtentorial gas is present beneath the frontal lobes. Gas is also seen in the lateral ventricles, in the fourth ventricle and in the basilar cisterns. The cerebral angiography was done about fifteen minutes after the pneumoencephalography had been completed.

of gas beneath the tentorium, and a small amount had entered the right lateral ventricle (Fig. 1*A*). The study was continued after the needle had been reinserted two interspaces higher, whereupon the gas entered the ventricular system disclosing a left parasagittal tumor (Fig. 1*B*).

In the second case the gas entered the subtentorial and subdural spaces (Fig. 2, *A* and *B*). The needle was withdrawn and inserted two interspaces higher. Injection of additional gas outlined the ventricular system in its normal position, with very little gas visible in the subdural area (Fig. 2*C*). Inasmuch as this examination was within normal limits, it was decided to perform percutaneous angiography. The lateral angiogram in the brow-up position showed subdural gas gathered over the frontal lobes, while the subarachnoid gas outlined the ventricular system (Fig. 2*D*).

SUMMARY

A technique is suggested for diminishing the amount of gas introduced into the subdural space during lumbar pneumoencephalography.

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LATERAL MIDLINE LAMINAGRAPHY IN ENCEPHALOGRAPHY AND VENTRICULOGRAPHY*

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LAMINAGRAPHY in pneumoencephalography and ventriculography, first introduced in 1940 by Lutz and Turner,[4] is used rather infrequently.[1,2,3] Many investigators consider the method cumbersome and time consuming. As one's technical experience grows, it is found possible for an efficient technician to set up the device and take one exposure in three minutes. In our department this leads to frequent use of the method as an aid in general roentgenographic diagnostic work.

Upon a few occasions patients suspected of having a posterior fossa tumor were proved by laminagraphy to have normal aqueducts and fourth ventricles (Fig. 1). With each such case our enthusiasm grew. It was, therefore, decided to employ the procedure routinely on all air studies for a period of time, to study the findings and re-assay their diagnostic value.

PHYSICAL CONSIDERATIONS

Many reports of the basic principles of laminagraphic devices are available and will not be discussed here. We operate a General Electric Ordograph at a 40 inch target-film distance, using a 15 inch arc of tube movement. This produces a depth of sharpness of 1.2 mm. which may permit taking a lateral midline laminagram with a single exposure.

Visualization of the third ventricle, aqueduct of Sylvius, and fourth ventricle by lateral midline laminagraphy has usually been accomplished in cases having dilated ventricles.[1–4] From such reports one may incorrectly assume that normal sized third ventricles and aqueducts cannot be readily visualized by this procedure.

To disprove this a pressdwood phantom of three different sized third ventricles and an aqueduct of Sylvius was constructed and the detectability of these structures studied (Fig. 2). The column of silhouettes indicates the cross-sectional shapes of the artificial third ventricles having transverse diameters of 13 mm., 9 mm. and 6 mm., respectively, and of an aqueduct of 2 mm. in diameter. The lower portions of the ventricles were made angular to simulate a small amount of residual cerebrospinal fluid. In *A* are shown the corresponding appearances in a conventional lateral roentgenogram of the phantom 15 cm. thick or comparable to the average skull thickness, and in *B* the corresponding appearances in the lateral laminagram of the phantom. In comparing the film strips, attention should be paid only to the upper and lower margins of the air-filled structures. The following conclusions are warranted: 1. Laminagraphy yields consistent improvement in detail regardless of size of the air-filled structures. 2. Laminagraphy obliterates the mottled effect of the superimposed pressdwood or in a patient it eliminates the undesirable shadows of bone, ear and extraneous air. 3. The lower margins of all the artificial ventricles are unsharp, regardless of the size, because of their angularity. This corresponds to a small volume of residual fluid in the patient. Therefore, in positioning the head of the patient (Fig. 3) the point of maximum interest should be elevated slightly, *i.e.*, for the aditus of the aqueduct, the brow and vertex should be closer to the table top than the chin and occiput. Figure 4 illustrates how much better a third ventricle having a transverse diameter of 2

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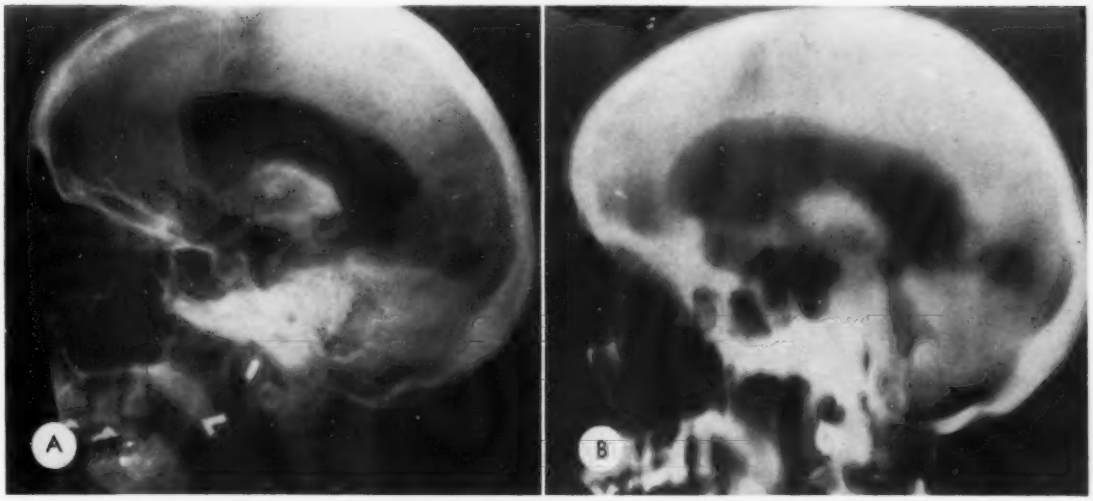

Fig. 1. (*A*) Conventional lateral pneumoencephalogram made with the brow down in a patient with dilated lateral and third ventricles. (*B*) Lateral laminagram adding improved visualization of aqueduct and fourth ventricle. The diagnosis was communicating hydrocephalus.

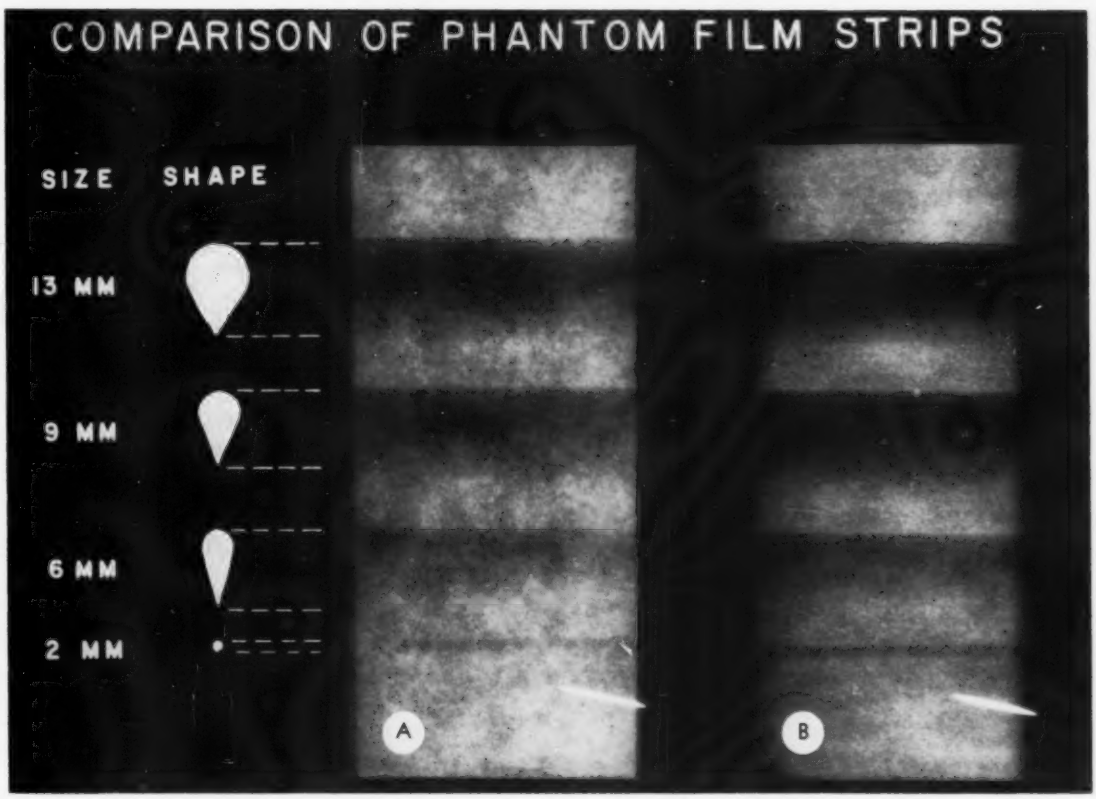

Fig. 2. Comparison of phantom film strips. (*A*) Roentgenogram. (*B*) Laminagram.

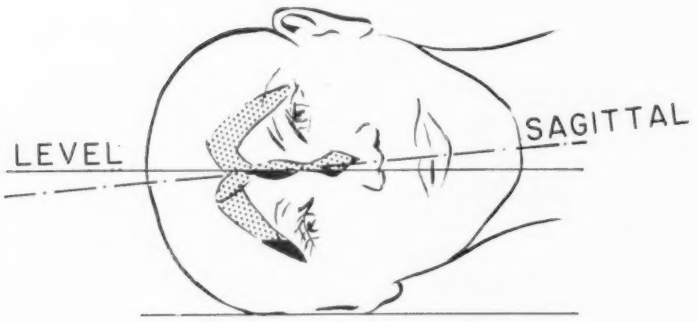

FIG. 3. Diagram of head position.

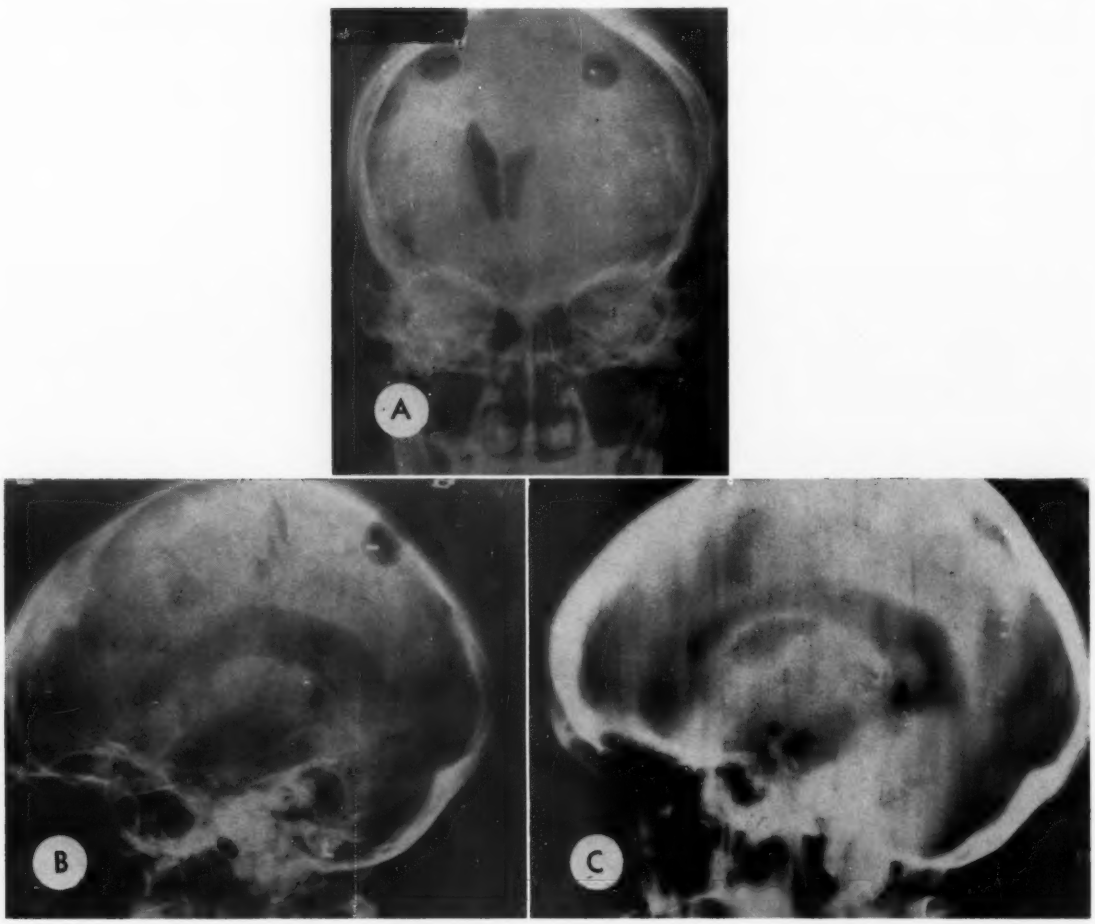

FIG. 4. (*A*) Posteroanterior pneumoencephalogram having a displaced 2 mm. third ventricle. (*B*) Conventional lateral view without visualization of the midline structures. (*C*) Lateral laminagram showing partial visualization of the third ventricle and aditus. The faintly calcified pineal gland is now seen to be displaced posteriorly. This and the blunted frontal horn was an aid in the diagnosis of a right frontal tumor. The fourth ventricle is normal.

Table I

	Not Adequately Visualized	Adequately Visualized
Third Ventricle		
Roentgenography in Conventional Views	15 cases	9
Laminagraphy	8	16
Aqueduct of Sylvius		
Roentgenography in Conventional Views	20	4
Laminagraphy	7	17
		If only the aditus was visualized the case was included here
Fourth Ventricle		
Roentgenography in Conventional Views	15	9
Laminagraphy	4	20

mm. can be visualized by laminagraphy than by roentgenography in conventional views.

Clinical Comparison of Roentgenograms in the Conventional Views and Lateral Midline Laminagrams

Lateral midline laminagrams were reviewed in 24 cases having had pneumoencephalography or ventriculography performed. These were graded on the basis of "not adequate" or "adequate" in reference to visualization of the third and fourth ventricles and aqueduct of Sylvius. The results are given in Table 1.

In pneumoencephalography, good filling did not always result in good conventional lateral roentgenograms of the third ventricle because of camouflaging air in the temporal horns, cysterna ambiens and cortical subarachnoid spaces.

In grading the visualization of the aqueduct, visualization of the aditus alone was considered "adequate visualization." Several aqueducts definitely demonstrated on the laminagrams were poorly delineated on the conventional lateral roentgenograms and often the soft tissue shadow of the ear produced a false shadow of the aqueduct and floor of the fourth ventricle.

The findings in reference to the fourth ventricle have been particularly valuable. By adding laminagraphy to our routine procedures we have been able to adequately demonstrate the fourth ventricle in all but 5 cases in this series. One of these was in the group of 7 cases adequately shown by conventional roentgenography. Two were due to non-filling of the ventricles. The remaining 2 cases in which the fourth ventricle was not visualized by laminagraphy were found to have pathologic conditions and are here illustrated. The first of these is shown in Figure 5. The abrupt ending of the air in the aqueduct without abnormal displacement of the aqueduct in a ventriculogram suggested the correct diagnosis of aqueduct stenosis. Another patient with an unfilled fourth ventricle was found to have a dilated aditus and nonfilling below this point (Fig. 6). The aditus was abnormally close to the dorsum of the

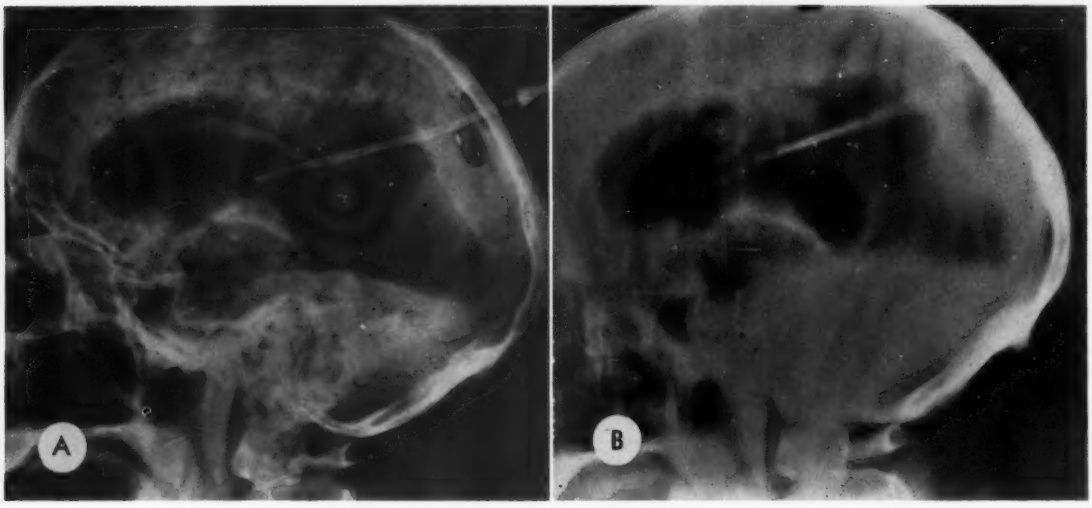

FIG. 5. (*A*) Conventional lateral ventriculogram with good filling of the third ventricle, but indistinct detail posteriorly because of temporal horn air. (*B*) Lateral laminagram showing cut off aqueduct. Lack of deformity of third ventricle and lack of displacement of aditus should suggest the correct diagnosis, aqueduct stenosis.

sella turcica and there was a prominent indentation of the peduncles on the third ventricle. This suggested a cerebellar tumor and the patient was found to have an astrocytoma at surgery.

CONCLUSION

1. The value of lateral midline laminagraphy in the study of the third ventricle, the aqueduct of Sylvius and fourth ventricle is re-emphasized.

2. Special attention should be given to positioning of the head to best visualize the third ventricle.

3. Since adding lateral laminagraphy to the routine air studies, the fourth ventricle has been demonstrated in all but 2 of 22 normal cases. The impression has been

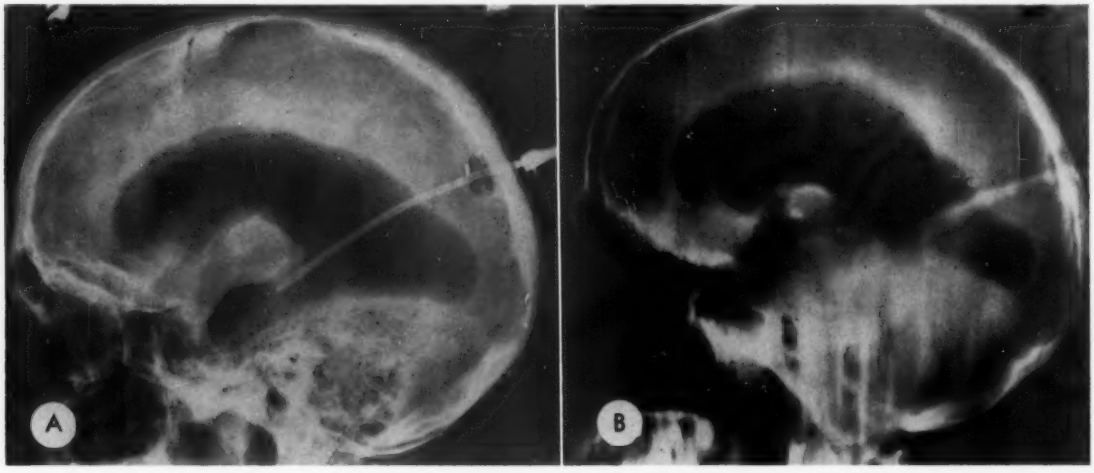

FIG. 6. (*A*) Conventional lateral ventriculogram with well filled third ventricle, but not well visualized because of large temporal horns. (*B*) Laminagram obliterates temporal horn shadows and renders full outline of third ventricle. The third ventricle is enlarged in the ventrodorsal dimension, but not above the upper limits in height. The aditus is abnormally close to the posterior clinoid of the sella turcica. The upper portion of the aqueduct is dilated to 4 mm. and cut off. The findings support a diagnosis of cerebellar tumor. At surgery a cerebellar astrocytoma was found.

gained that if there is a moderate amount of ventricular filling and the fourth ventricle is not shown by laminagraphy, a cerebellar or brain stem lesion may be suspected.

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MYELOSCINTIGRAPHY: A USEFUL PROCEDURE FOR LOCALIZATION OF SPINAL BLOCK LESIONS*

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THE use of radioactive iodinated human serum albumin (RIHSA) in localizing lesions of the spinal canal has been described by Bauer and Yuhl[1] in a preliminary report. Since June, 1955, we have employed this technique for localization of lesions which show clinical evidence of spinal cord or root compression and varying degrees of spinal block. Our neurosurgical department has found this to be a useful procedure in more accurately identifying the level of spinal block lesions.

The technique we have used is similar to that outlined by Bauer and Yuhl.[1] If, at the time of the usual diagnostic lumbar spinal puncture there is evidence of a partial or complete spinal block, the isotope laboratory is called and approximately 300 μc of RIHSA in 5 cc. of normal sterile saline is injected before the spinal needle is withdrawn. If possible, the patient is placed in the knee-chest position for at least fifteen minutes. If not, the foot of the bed is elevated. One hour after injection, the patient is scanned, using a commercially available, mechanical apparatus (Reed-Curtis scintiscanner). We feel that this equipment is already somewhat outmoded (Fig. 1, *A* and *B*).

The scintigrams obtained are referred to as myeloscintigrams. The myeloscintigram is orientated as to level by means of a lead marker placed on the patient's back, its location being identified on the scintigram. A roentgenogram of the involved area is made with the lead marker still in position. The scintigram is then superimposed upon the roentgenogram for localization of the spinal block (Fig. 2).

It is important to note that the recorded activity may normally "fade out" at the lower end of the spinal subarachnoid space. The exact level of this fade out depends upon the size and position of the caudal sac. If a low lesion is suspected, it is also advisable to elevate the patient to a sitting position for several minutes prior to scanning.

It has been shown that within twenty-four hours 40 to 50 per cent of the RIHSA will have left the cerebrospinal fluid.[1,2] Most of the I^{131} will remain bound to the

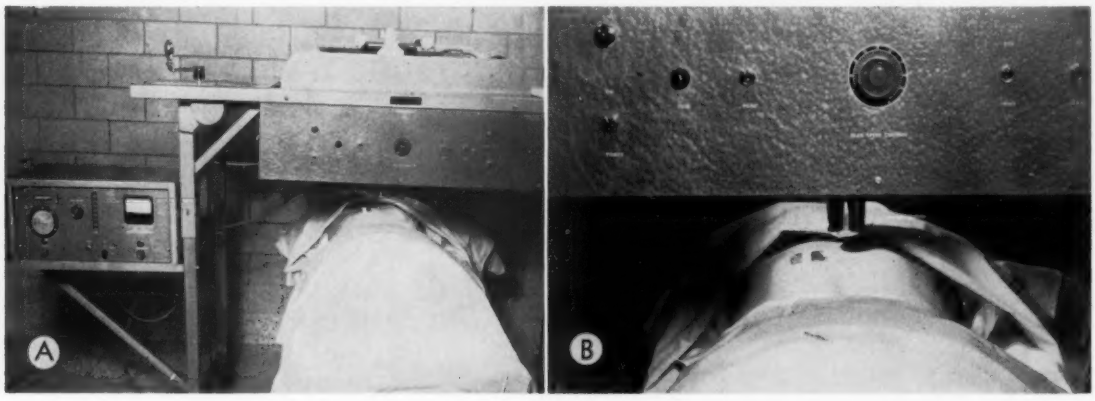

FIG. 1. (*A*) Patient and scanner prepared for scanning. (*B*) Note lead marker taped to patient's back. This is used as reference in orienting the scintigram with a roentgenogram of the spine.

* From the Departments of Radiology and Neurosurgery, Mercy Hospital, Pittsburgh, Pennsylvania. Presented at the Fifty-seventh Annual Meeting of the American Roentgen Ray Society, Los Angeles, California, September 25–28, 1956.

protein.[1,2] Only approximately 5 per cent will be picked up by the thyroid. This small amount of thyroid uptake of I^{131} may be prevented by the administration of Lugol's solution. It is of course desirable to begin administration of Lugol's solution twenty-four hours before the injection of RIHSA. In those instances in which the severity or rapid progress of the neurologic findings has made early surgical intervention imperative, we have not considered the twenty-four hour period necessary. In fact, there is good reason to believe that such a delay is not required for adequate blocking of the thyroid. We have observed in the determination of thyroid uptake of I^{131} that the accidental administration of one dose (10 drops) of Lugol's solution, or four tablets (2.0 gm.) of telepaque as late as six to eight hours after the administration of I^{131} (sodium radioiodide) may reduce the thyroid uptake to 2 to 8 per cent of the administered dose.

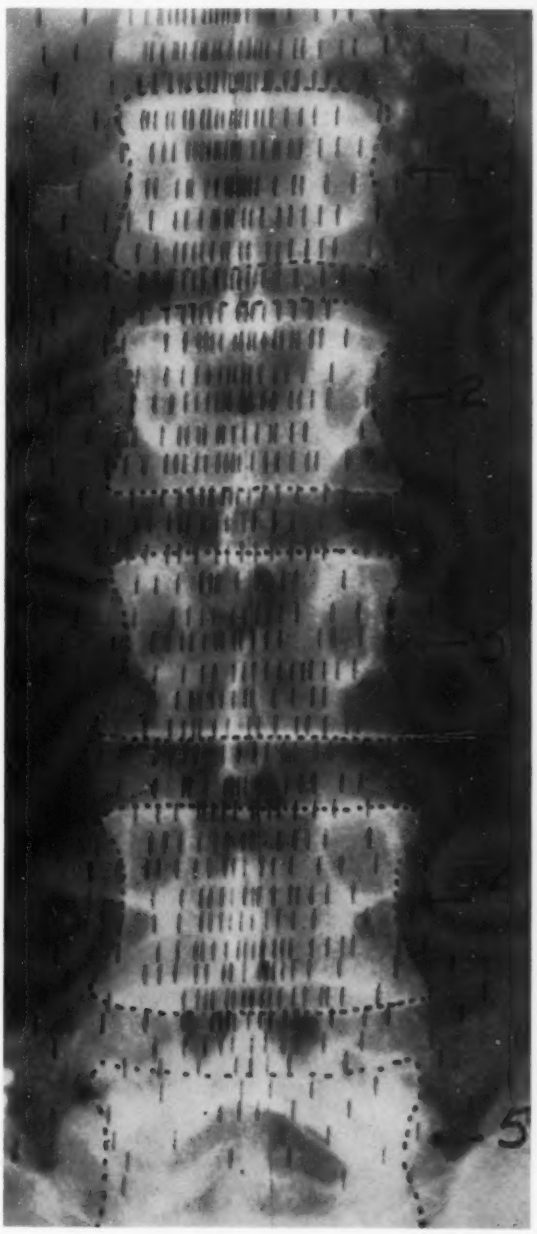

Fig. 2. Normal lumbar myeloscintigram.

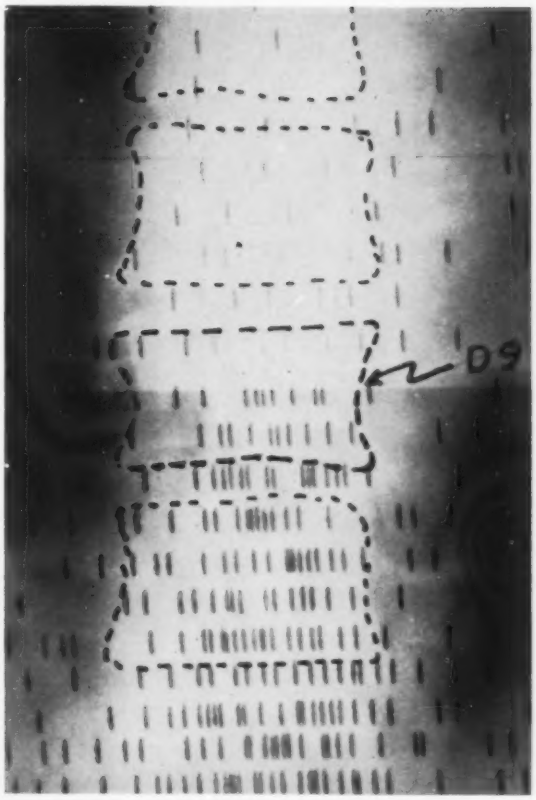

Fig. 3. Case 1. (G.M.) Complete block at the upper margin of D-9.

We have noticed no significant untoward reaction in 28 patients subjected to this procedure.

The early cases in this series did not all have clinical evidence of a spinal block but they are included for the sake of completeness. At the present time the technique is usually reserved for those indi-

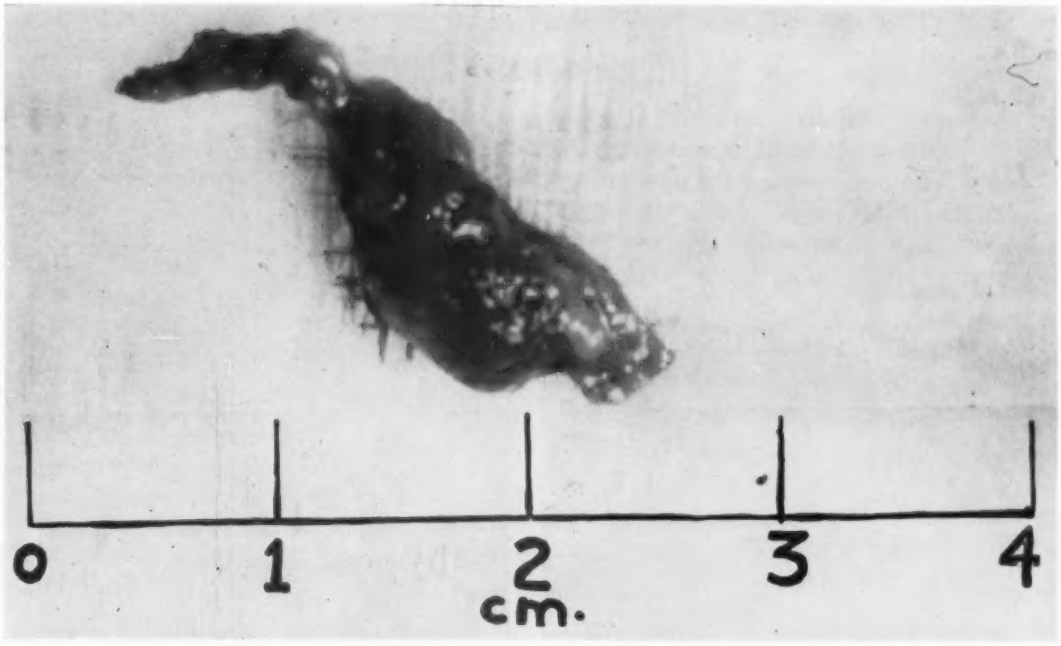

FIG. 4. Case I. (G.M.) Arachnoid cyst found at lower margin of D-8.

viduals with clinical evidence of either partial or complete spinal block.

REPORT OF CASES

CASE I. (G.M.) This forty-seven year old white male was admitted to the hospital because of pain in the back and left side of the abdomen. This pain had developed suddenly ten days prior to admission. Neurologic examination indicated a spinal cord lesion near D-9. The spinal block test was positive. A confirmatory myeloscintigram demonstrated com-

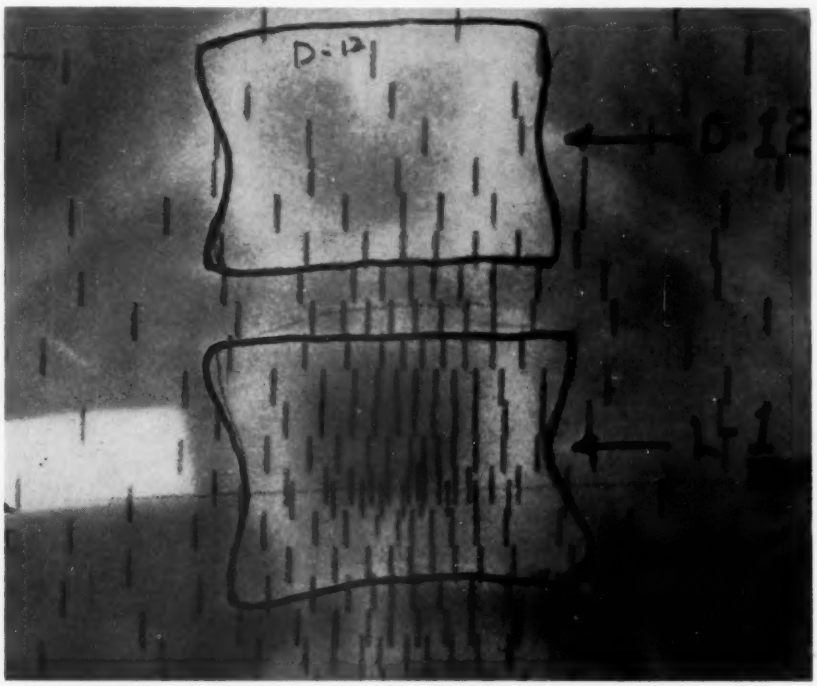

FIG. 5. Case II. (R.L.) Complete block at the lower margin of D-12 due to an epidural abscess.

plete block at the upper margin of D-9 (Fig. 3). Laminectomy revealed a mass lesion on the left at D-8. This was removed and proved to be an arachnoid cyst (Fig. 4).

Case II. (R. L.) This patient was an eighteen year old white male, a known diabetic. One week before admission he had injured his back while performing gymnastics but had been able to continue routine activities. Three days prior to admission the pain became more severe and was followed rapidly over the next twenty-four hours by weakness, loss of sensation and finally paralysis of both lower extremities. The spinal block test was positive. A myeloscintigram showed a complete block at the lower margin of D-12 (Fig. 5). Laminectomy demonstrated an epidural abscess at this level.

Case III. (M.C.) This twelve year old white female was admitted with complete paraplegia. She had complained of intermittent backache for one year. During the six weeks immediately

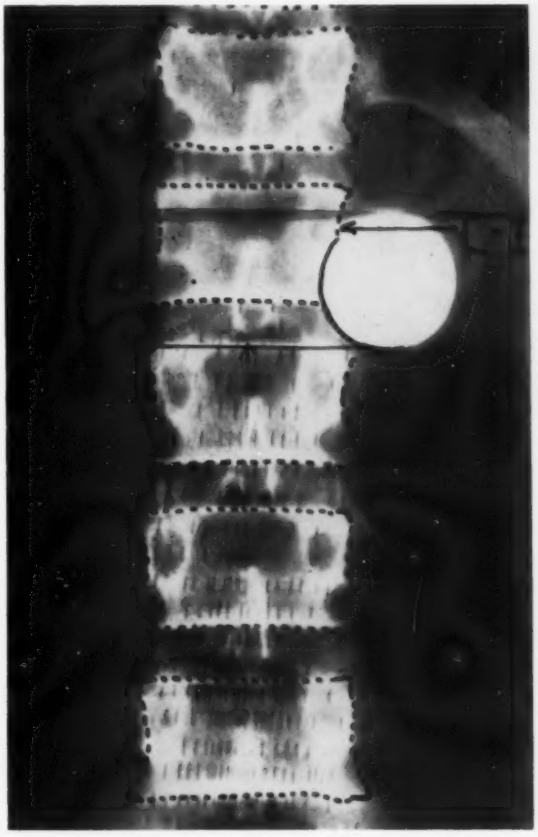

Fig. 6. Case III. (M.C.) Complete block at the first lumbar interspace due to an epidural lymphosarcoma.

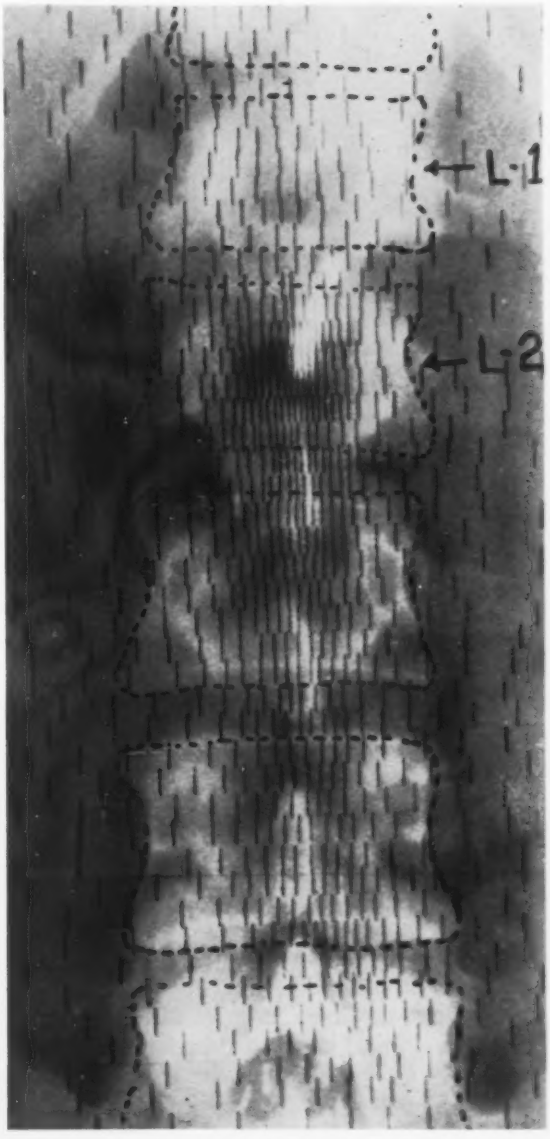

Fig. 7. Case IV. (R.S.) Incomplete block at the first lumbar vertebra due to a meningioma.

before admission this had progressed in severity and frequency. She had received six treatments by a chiropractor without relief and followed by sudden onset of total paralysis of her legs. Neurologic findings were considered to indicate a lesion at L-3 or L-4. The spinal block test was positive and a myeloscintigram demonstrated a complete block at the first lumbar interspace or superior margin of L-2 (Fig. 6). Laminectomy revealed an epidural lymphosarcoma at the upper border of L-2 extending to the upper border of D-12.

Case IV. (R.S.) This fifty-five year old white

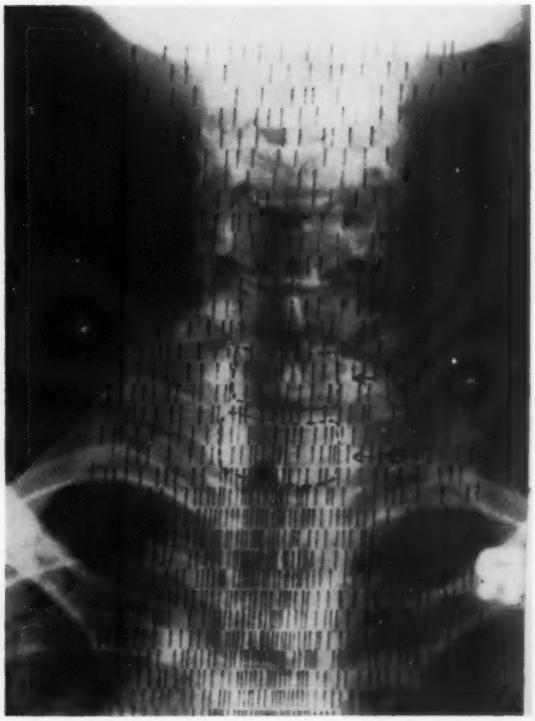

FIG. 8. Case v. (E.L.) Incomplete block at the level of C-6–C-7 on the left side, due to a meningioma.

female was admitted because of constant pain in the right buttock of nine months' duration. There was radiation of the pain down the right thigh and aggravation of the pain by cough and deep inspiration. The clinical impression was a space occupying lesion at L-4. Lumbar puncture revealed evidence of an incomplete spinal block. A pantopaque myelogram done at another hospital had been interpreted as normal. A myeloscintigram was interpreted as demonstrating an incomplete block at L-1 (Fig. 7). Laminectomy revealed a meningioma at this level.

CASE V. (E.L.) This fifty-two year old white female was admitted to the hospital complaining of difficulty in walking of one year's duration followed by numbness of the left hand. Neurologic examination suggested a lesion at the level of C-7 or D-1. A myeloscintigram demonstrated a space occupying lesion at C-6 and C-7 on the left side producing an incomplete block (Fig. 8). Laminectomy revealed a meningioma arising at the level of the sixth cervical nerve root on the left side.

CASE VI. (A.H.) This thirty-six year old white male complained of low back pain radiating into the left leg following a fall several months prior to admission. On the morning of admission to this hospital, he stated that after bending over he had severe exacerbation of the back pain. Neurologic examination suggested a lesion at L-5 on the left side, but because of the lack of motor and sensory disturbance, a myeloscintigram was performed. This indicated a lesion at the L-4 intervertebral space (Fig. 9). A pantopaque myelogram revealed an incomplete block of the lumbar canal at the L-4 interspace (Fig. 10). A modified laminectomy was performed for removal of a massive ruptured disk at the L-4 interspace.

DISCUSSION

Of the 28 patients studied thus far, 12 myeloscintigrams were reported as show-

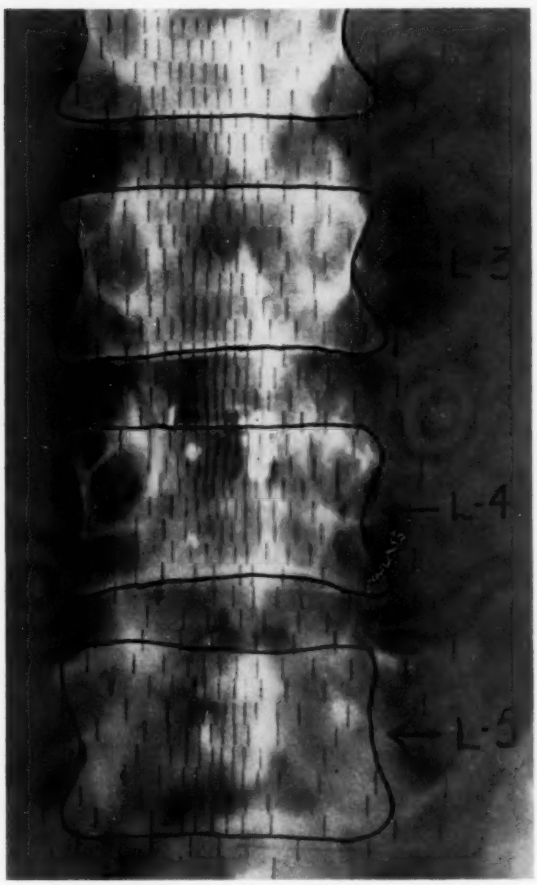

FIG. 9. Case VI. (A.H.) Nearly complete block at the fourth lumbar interspace due to a massive, bilateral ruptured intervertebral disk.

ing positive evidence of an abnormality such as complete block, partial block, or interference with the circulation of the spinal fluid. Ten such reports were verified by operation. One was reported as showing a filling defect at the fourth lumbar interspace, but laminectomy showed a herniated intervertebral disk at the fifth lumbar intervetebral space. The twelfth positive case was clinically diagnosed arachnoiditis and not operated upon.

Of 9 cases reported as negative, 2 were found to have small herniated disks at operation. One had diffuse arachnoiditis and the remaining 6 were not operated upon.

Seven myeloscintigrams were reported as uncertain. Six of these were considered to be suggestive of a spinal lesion. Three of the latter were operated upon and shown to have a lesion at operation. In the remaining 4, clinical evidence was not considered sufficient to warrant operation.

Fig. 10. Case VI. (A.H.) Pantopaque myelogram; lateral view demonstrating block at L-4 interspace.

Fig. 11. Myeloscintigram of a patient with extensive arachnoiditis confirmed by laminectomy. Note irregular pattern at L-2 and L-3.

It is of special interest to note that in the 9 proved cases with evidence of partial or complete block at the time of spinal tap, the myeloscintigram localization was correct in all, or 100 per cent of the cases. It is in these cases that we believe our myeloscintigraphy is of greatest value.

In some cases, neurologic localization has been uncertain or incorrect despite the presence of a partial block. Case IV is an excellent example of this with clinical localization at the level of L-4 and myeloscintigram localization of a meningioma at L-1 (Fig. 7).

Clinical determination of the lowermost limits of elongated spinal canal lesions is not infrequently difficult. In Case III, neu-

TABLE I

TABULATION OF CASES

Patient	Spinal Block Test	Clinical Level	Myeloscintigraphic Level	Operative Findings (Level of Lesion)
I. G.M.	Positive	D-9	D-9, upper margin	D-8 (arachnoid cyst)
II. R.L.	Positive	Not stated	D-12	D-12 (epidural abscess)
III. M.C.	Positive	L-3 or L-4	L-1 interspace, lower limit	L-2 extension to D-12 (lymphosarcoma)
IV. R.S.	Partial	L-4	L-1	L-1 (meningioma)
V. E.L.	Positive	C-7–D-1	C-6–C-7 on left side	C-6–C-7 (Meningioma on left side)
VI. A.H.	Not done	Uncertain L-5	L-4 interspace, positive	L-4 interspace (bilateral disk)
VII. W.H.	Negative	C-5 or C-6	Uncertain	C-5–C-6 (Bony ridge)
VIII. W.W.	Positive (2 tests)	Indefinite	D-8–D-5, no block, gradual decrease from this level	Arachnoiditis
IX. J.C.	Not reported	None	Negative	No operation
X. H.E.	Not done	L-4 or L-5	Negative	L-4–L-5 (disk)
XI. S.M.	Negative	Indefinite	Negative	No operation
XII. E.P.	Not done	None	Negative	No operation
XIII. J.P.	Negative	L-3 or L-4	Negative	L-3 (disk)
XIV. M.E.	Not reported	L-5	Uncertain	L-5 (disk)
XV. J.H.	Not done	D-12–L-1	D-12, upper half	D-11 (dermoid)
XVI. M.M.	Not done	None	Negative	No operation
XVII. G.U.	Partial (?)	Indefinite	Negative	No operation
XVIII. J.S.	No record	L-5	Negative	L-5 (disk)
XIX. H.H.	No record	Indefinite L-3, 4 or -5	Negative	L-4 (varicosities)
XX. B.C.	Not done	Indefinite	Negative	No operation
XXI. W.W.	Negative	None	Negative	No operation
XXII. D.W.	Positive	D-11–D-12	D-10–D-11, block	D-11 (bilateral disk)
XXIII. J.W.	Positive	L-4 or L-5	L-4, middle of body	No operation (arachnoiditis)
XXIV. J.B.	Negative	None	D-12–L-1, questionable block	No operation
XXV. G.M.	Indefinite partial block	L-3 or L-4	L-4, block	L-4 (adhesive arachnoiditis)
XXVI. Sr. M.P.	Partial	Indefinite	No definite block ?defect	No operation
XXVII. H.W.	Negative	Indefinite	D-10–D-11, no block, decreased believed from this level	No operation
XXVIII. M.C.	Negative	Indefinite	D-12, partial obstruction	No operation

rologic findings indicated a lesion at L-3 or L-4 and the myeloscintigram was of definite value in demonstrating the lower margin of the lymphosarcoma to be at the first lumbar interspace (Fig. 6). It is readily apparent that, as with contrast myelography, more accurate localization is helpful to the surgeon, enabling him to limit his operative field and eliminate unnecessary exploratory incisions.

Upon comparing the myeloscintigrams of several of the patients believed to have arachnoiditis, we have noted that in the area of involvement there is irregular distribution of radioactivity or a gradual diminution over a segment with a more

normal pattern above and below the involved area (Fig. 11).

It is therefore felt that myeloscintigraphy may ultimately prove to be of value in helping to establish the diagnosis of inflammatory as well as space occupying lesions of the spinal cord and canal. Further investigation of this possibility is at least indicated.

Three errors were made on interpretation of the myeloscintigrams of patients with small herniated intervertebral disks. We believe this emphasizes the limitations of the present technique, indicating that the lesions must be large enough to interfere with spinal fluid circulation in order to be *consistently* demonstrated. There were two instances of correct positive localization of large bilateral herniated intervertebral disks.

Improvement in the "resolving power" of the scanning apparatus probably offers the best means of increasing accuracy in localizing lesions that do not produce a block of the subarachnoid pathway. The photoscanning techniques reported by Kuhl, Chamberlain, Hale and Gorson,[5] and by Bender[2] show promise of greatly improving myeloscintigraphic results. In addition, improved collimators may further enhance the results.

The simplicity of the technique makes it highly desirable and in this manner somewhat comparable to air myelography. Camp,[3] in an excellent article on contrast myelography, has enumerated the advantages and disadvantages of air myelography, and it would appear in our hands, at least, that myeloscintigraphy has fewer side reactions and will prove to be more satisfactory for the demonstration of partially obstructing lesions.

The average time required for scanning has been twenty-five minutes when 300 μc were used. Since the RIHSA is rather rapidly eliminated from the spinal fluid and excreted, there is no necessity to or difficulty in removing it as there is with potentially irritating radiopaque materials. The patient is, of course, much more comfortable during this procedure than during oil contrast myelography and there is no need for fluoroscopy.

SUMMARY

Twenty-eight myeloscintigraphies have been performed on patients with spinal lesions. Analysis of the series shows that the procedure has been accurate in localizing all lesions large enough to produce partial or complete spinal block. Smaller lesions are not consistently localized with our present equipment.

The procedure is simple, easily performed and produces no undesirable side effects. The useful scope of myeloscintigraphy may be widened with improved equipment.

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PHYSICAL FACTORS WHICH DETERMINE ROENTGENOGRAPHIC CONTRAST*

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TWO physical factors determine the visibility of images in a roentgenogram, *viz.*, *contrast* and *definition*. They are recognized as of equal importance but only contrast is treated in this paper. The relationship between contrast, definition, and visibility of detail is qualitative, not quantitative. An image having high contrast, that is, one in which density differences are large but boundaries are unsharp, may be of equal visibility to one in which the contrast is low and the boundaries sharp. Thus, for equal visibility, there is a reciprocal relation between contrast and definition, but, since the two are not the same *kind* of quantity, a proper comparison cannot be made.

The contrast in a roentgenogram is the result of the combined effects of roentgen-ray absorption, roentgen-ray scattering, and the contrast of the film itself. Exposing a roentgenographic film to make a roentgenogram has its analogue in the exposing of a film in a camera, a process which is more familiar to us in the physical sense because visible light effects are more deeply a part of common experience than are roentgen-ray effects.

(1) Roentgen-ray absorption is directly responsible for the variation in roentgen-ray intensities transmitted by various body parts and may be likened photographically to the variation in reflection of light from the different objects in a scene.

(2) Roentgen-ray scattering acts roentgenographically as light scattered by fog acts photographically. The unsatisfactory result in a photograph made on a foggy day is not caused by insufficient light, for this can be corrected by suitable exposure if necessary, but by the way in which the illumination due to the fog alters the *relative* brightnesses in the scene.

(3) The contrast of a roentgenographic film determines the contrast in the final image in just the same way as does a photographic film. In general, roentgenographic films are of such high contrast that the relative brightnesses in a roentgenogram as viewed on an illuminator are greater than the relative brightnesses of the intensifying screen which exposed the film. Thus the roentgenographic film is a "contrast amplifier."

Roentgen rays are absorbed according to the Equation,

$$I = I_0 e^{-\mu x}, \qquad (1)$$

in which I is the intensity of the beam transmitted by a thickness x of material on which the beam I_0 is incident. The absorption coefficient μ depends on the density and atomic number of the material and on the wavelength of the roentgen rays. This equation applies strictly only to monochromatic roentgen rays and for conditions of no scattering. The equation is essential in the present discussion but it can be used for calculations in roentgenographic problems only with caution because the broad roentgen-ray spectrum is continuously modified by preferential absorption of the longer wavelengths as the beam passes through the patient and no one value of μ can strictly be applied. Further, very special geometric arrangements are necessary to avoid errors due to scattering.[4,5] For absorption data applicable to roentgenographic conditions, the reader is referred to Morgan.[2]

SUBJECT CONTRAST

A quantity called subject contrast can be defined as the ratio of the roentgen-ray intensity through a certain part to that through an adjacent, more absorbing part

* Communication No. 1908 from the Kodak Research Laboratories.

(see Fig. 1). If roentgen rays fall on two different thicknesses of tissue, the thinner transmits an intensity I_0 and this is also the intensity which falls on the *additional* thickness x of the thicker part, which then transmits an intensity I. The ratio I_0/I will, by definition, be greater than unity because I_0 always represents the roentgen-ray intensity through the thinner or less absorbing part.* If one thickness of tissue is *much* greater than the other, I_0/I will be larger than if the two are nearly the same.

It follows from Equation (1) that

$$\frac{I_0}{I} = e^{\mu x}; \tag{2}$$

but to apply this to the problem of subject contrast, the thickness x, in the case of tissues referred to above, is the *difference* in the two thicknesses (Fig. 1). It is apparent from Equation (2) that as x grows larger, with μ remaining constant, I_0/I increases. Thus, the greater the thickness difference, the greater is the difference in relative roentgen-ray transmission.

Since I_0/I depends on the product of μ and x, an increase in μ (lowering kilovoltage) could be compensated for by a decrease in thickness difference, thus keeping I_0/I the same. This can be demonstrated experimentally, though a trial-and-error procedure would be used to find the kilovoltage at which matching roentgenograms would be obtained.

In preceding paragraphs, a quantity called subject contrast was defined which was symbolically expressed as I_0/I. Considering roentgen-ray absorption only, $I_0/I = e^{\mu x}$. This is an oversimplification of the roentgenographic problem because roentgen-ray scattering, which may be very great, has been completely ignored.

Scattered roentgen rays, like the light scattered by fog, are quite diffuse. In the roentgen-ray image, therefore, the amount of scattered roentgen rays added to adjacent areas is about the same, even though the absorption of the primary beam is different in the two areas. This means that subject contrast, originally expressed as I_0/I, now becomes

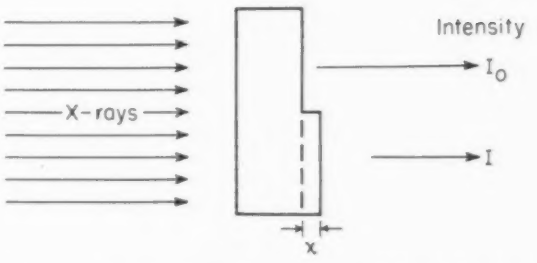

Fig. 1. A homogeneous piece of material with thickness difference x transmits roentgen rays of intensities I and I_0 through the thick and thin parts, respectively.

$$\frac{I_0 + I_s}{I + I_s} = C_s, \tag{3}$$

where I_s is the intensity of scattered radiation and C_s is subject contrast *with scatter included*.[6] In roentgenography of the human chest, the scattered radiation is about equal to the primary, image-forming roentgen rays,[8] but in roentgenography of the abdominal region, with its much greater amount of matter, the scatter is several times greater. From laboratory measurements, it has been found that the proportion of scattered radiation in roentgenography may be as high as 10 or more,[3,7,9] assuming that the primary, image-forming radiation is 1; *i.e.*, there may be more than 10 times as much scattered radiation as primary radiation.

From Equation (2), $I_0 = Ie^{\mu x}$ and this value of I_0 may be substituted in Equation (3), giving:

$$Ie^{\mu x} + I_s = C_s(I + I_s). \tag{4}$$

As the ratio I_s/I, that is, the proportion of scattered radiation to image-forming radiation, is of significance rather than the separate quantities, I_s and I, Equation (4) is put in a more convenient form by dividing through by I, giving

$$e^{\mu x} + \frac{I_s}{I} = C_s\left(1 + \frac{I_s}{I}\right)$$

* For some purposes, subject contrast may be defined as $\log_{10} I_0/I$ but this does not alter the basic concept.

or

$$C_s = \frac{e^{\mu x} + \frac{I_s}{I}}{1 + \frac{I_s}{I}}. \qquad (5)$$

Equation (5) thus gives the subject contrast in terms of both the absorption and the proportion of scattered radiation.

FILM CONTRAST

Regardless of how the roentgen rays are absorbed and scattered in the patient, the net result is an image which is to be recorded on film. The contrast characteristics of the film must therefore be considered.

Film contrast may be defined for quantitative purposes as the slope $\overline{G}$ of the straight line drawn between specified densities on the characteristic curve. This is called "average gradient."[1] The density limits of the interval may be chosen so as to be most appropriate for the problem at hand but they must be specified in order that $\overline{G}$ shall have complete quantitative significance.

The defining equation for average gradient is

$$\overline{G} = \frac{D_2 - D_1}{\log_{10} E_2 - \log_{10} E_1}. \qquad (6)$$

See Figure 2.

The densities D_2 and D_1 correspond to exposures E_2 and E_1, respectively. Photographic exposure $E = It$, where I and t are intensity and time, respectively. The exposure in different parts of a roentgenogram differs because of the various intensities but with the time of exposure constant.

Subject contrast, as already explained and illustrated by Figure 1, is the ratio of two intensities. If these two intensities are designated I_1 and I_2 to conform to the subscripts used in Equation (6), subject contrast is given by

$$C_s = \frac{E_2}{E_1} \quad \text{for} \quad E_2 = I_2 t \quad \text{and} \quad E_1 = I_1 t,$$

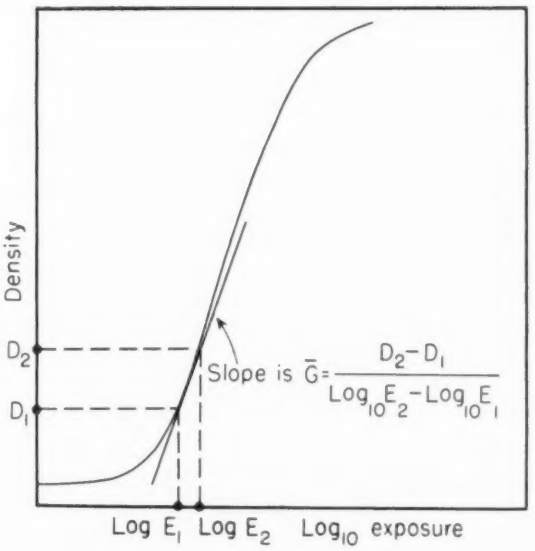

Fig. 2. Characteristic, or H and D curve of a roentgenographic film exposed to screen light, illustrating the definition of average gradient $\overline{G}$.

where t is the time of exposure. Therefore,

$$\log_{10} C_s = \log_{10}\left(\frac{E_2}{E_1}\right) = \log_{10} E_2 - \log_{10} E_1. \qquad (7)$$

Substituting

$$\log_{10} C_s \quad \text{for} \quad \log_{10} E_2 - \log_{10} E_1$$

in Equation (6) gives

$$D_2 - D_1 = \overline{G} \log_{10} C_s. \qquad (8)$$

Substituting the value of C_s from Equation (5) gives

$$D_2 - D_1 = \overline{G} \log_{10} \left(\frac{e^{\mu x} + \frac{I_s}{I}}{1 + \frac{I_s}{I}} \right). \qquad (9)$$

The density difference, $D_2 - D_1$, in some part of the image in the roentgenogram is called the "roentgenographic contrast" for that area. The roentgenographic contrast is often spoken of as "high" or "low" in referring to the entire roentgenogram. What is meant is that density differences in restricted areas all over the roentgenogram are relatively high or low.

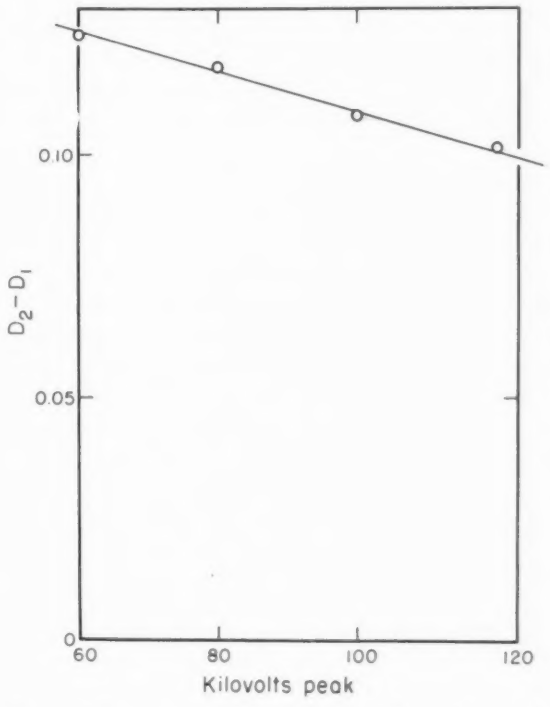

Fig. 3. Roentgenographic contrast for a piece of lucite plastic about 2 mm. thick as a function of roentgen-ray tube potential.

There are combined in Equation (9) all of the principal factors which determine the contrast in a roentgenogram. Roentgen-ray absorption is represented by $e^{\mu x}$. It is governed by the thickness difference x as well as by μ which, in turn, is affected by the kind of material, *e.g.*, flesh or bone. The kilovoltage applied to the roentgen-ray tube also affects μ because of the dependence of "average" wavelength on kilovoltage.

In Figure 3 is a plot of the roentgenographic contrast, D_2-D_1, against the kilovoltage applied to the roentgen-ray tube for a constant thickness of about two mm. of lucite plastic. The roentgen rays were pre-filtered by one-half inch of aluminum. Lucite plastic has roentgen-ray absorption characteristics similar to those of flesh. Scattered radiation was excluded, hence $I_s/I=0$ in Equation (9). These experimental conditions are roughly similar to those of some medical roentgenographic techniques except that scatter was absent. It is apparent that roentgenographic contrast diminishes with increasing kilovoltage, the latter being the cause of changes in μ in Equation (9).

The effect of the proportion of scattered radiation, I_s/I, can be most readily seen by examining Figure 4. With increasing amounts of scatter, the density difference, D_2-D_1, grows progressively smaller. Conversely, removal of scatter, *e.g.*, with a Bucky grid, leads to a rapidly increasing contrast, depending on the amount removed. Scattered radiation is thus seen to be a factor of primary concern to the radiologist since it directly affects what he is able to see in the roentgenogram. It is important to note that this radiation affects the roentgen-ray pattern "in space" before it strikes the cassette. In other words, the *film or film screen combination* does not distinguish between scattered and primary radiation.

The contrast effect of the film is all com-

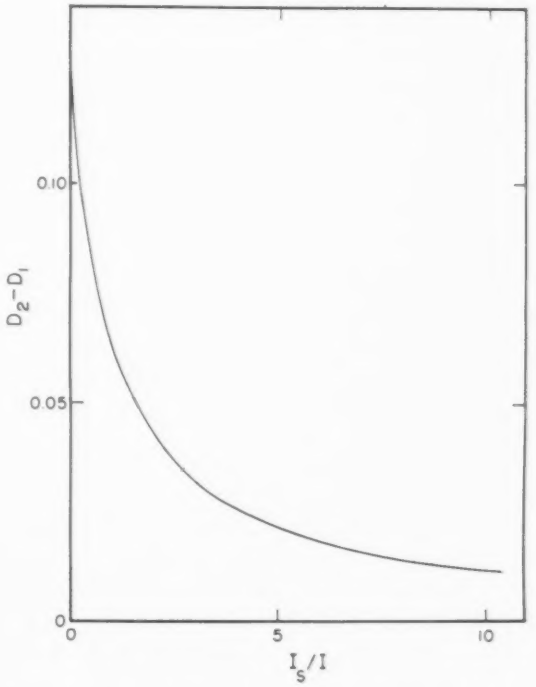

Fig. 4. Graph of Equation (9) with arbitrary values of 3.00 and 1.10 assigned to $\bar{G}$ and $e^{\mu x}$, respectively. Comparison of Figures 3 and 4 shows a fairly slow change in contrast with kilovoltage and a rapid change with proportion of scattered radiation.

bined in $\overline{G}$. Obviously, roentgenographic contrast is directly proportional to $\overline{G}$. In general, $\overline{G}$ increases with density in the toe of the H and D curve, then remains essentially constant, and finally decreases through the region of very high densities. Development time, temperature, and other processing factors also affect $\overline{G}$ in well-known ways. The low value of $\overline{G}$ in the toe of the curve accounts for the preference for moderately high densities in roentgenograms. If the H and D curve were a straight line, $\overline{G}$ would be constant throughout.

The foregoing discussion, leading to Equation (9), is a simpler treatment than that given by Morgan.[2] However, it may appeal to those who prefer brevity and an approximate treatment to one of more elegance. Another equation which has been published on this subject does not agree with Equation (9) but the derivation was not given so the origin of the discrepancy is not apparent.[3]

SUMMARY

The three factors which determine the contrast in a roentgenogram, *viz.*, roentgen-ray absorption, roentgen-ray scattering, and film contrast, are considered qualitatively and then mathematically. An equation is derived which shows their relationship.

The roentgenographic contrast of a tissue-like material changes comparatively slowly in the kilovoltage range common in diagnostic work. Scattered roentgen rays have a very pronounced effect on contrast. Roentgenographic contrast at a given density level is directly proportional to the average gradient of the film at that level.

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RECENT DEVELOPMENTS OF GLASS DOSIMETRY FOR SOFT ROENTGEN RAYS. PART I*

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DURING the past three years glass dosimetry has been studied intensively and has provided new and valuable contributions to radiation dosimetry. It is well known that glass exposed to ionizing radiations changes its optical properties, and two of these effects have been utilized for dosimetry: (1) The radiophotoluminescent response, which is useful for measuring small doses (personnel dosimetry),[9,10] and (2) the change in optical density, an effect which is well suited for measuring large doses.[2,5,6,8,11]

The second effect has been utilized only for high energy roentgen rays and electrons, and there is little information concerning its usefulness for soft roentgen rays. Due to the high absorption in the low energy range,[5,10] a high efficiency of the glass dosimeter may be expected. Radiation-induced changes in optical density have recently been investigated by the present authors[1] in experiments where commercially available glass coverslips were used for dose determinations.

The present study extends these investigations to problems which are specific for this energy range. They include: (a) The consequences of variations in absorbing glass thickness, and (b) the energy dependence of the glass dosimeter for various filtration of a 35 kev. roentgen-ray spectrum, with half-value layers (HVL) ranging from 0.1 to 1.3 mm. Al.

MATERIALS AND METHOD

In the biologic work, the glass dosimeter was used for 35 kev. roentgen rays from an AEG-50 Machlett roentgen-ray tube with 1 mm. beryllium window. The glass coverslip was used for delimiting the irradiation chamber for the biologic object. In order to minimize shielding the coverslip should be as thin as possible; therefore, for calibration purposes (Zn, Sb, Pb) activated sodasilicate glass of only 0.18 mm. thickness was used (chemical composition SiO_2 68.9 per cent, Na_2O 12.2 per cent, Al_2O_3 0.8 per cent, Sb_2O_3 6.3 per cent, PbO 6.7 per cent, ZnO 5.1 per cent).

The radiation induced colorization in the coverslip was measured as change in light transmission at 3250 Å, where the effect had been found to be most pronounced, and presented as changes in the linear absorption coefficient (μ), according to the following formula:

$$\mu = \frac{1}{T} \ln \frac{I_0}{I} \qquad (1)$$

where T is the thickness of the coverslip (0.18 mm.), I_0 and I are the light transmissions measured before and after irradiation.

The radiation induced colorization is an unstable effect, diminishing with time. The present authors have previously given a mathematical interpretation of this fading effect.[1]

Coverslips have been irradiated with doses ranging from $1.5.10^3$ to $1.4.10^7$ r. The resulting calibration curve is presented in Fig. 1.

If the results are replotted on probability paper, a straight line is obtained, which shows that the calibration curve represents the integral of a Gaussian curve. The mathematical relationship between the dose (D) and the linear absorption coefficient (μ) can therefore be expressed as

$$\mu = \frac{k}{\sigma\sqrt{2\pi}} \int_{-\infty}^{\ln D} \exp -\frac{(\ln D - \rho)^2}{2\sigma^2} \, d(\ln D) \qquad (2)$$

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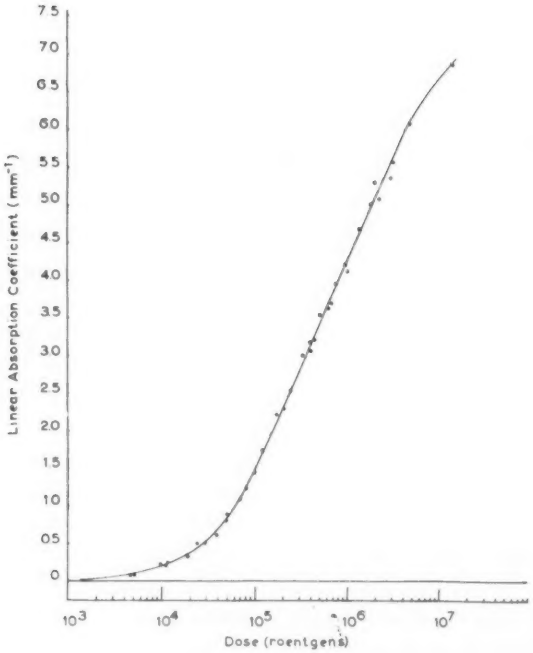

FIG. 1. Radiation induced linear absorption coefficient as a function of dose.

The numercial values of the parameters are $k=19.34$, $\sigma=2.21$, and $\rho=\ln 7.10^5$.

This signifies that $d\mu/dD \cdot D$ is normally distributed with a maximum at 7.10^5 r.

CONSEQUENCES OF VARIATIONS IN COVERSLIP THICKNESS

Due to the low energy radiation used, the color distribution through the coverslip is not homogeneous and decreases with depth according to a function $\mu(x)$. Experiments carried out by irradiating stacks of coverslips showed that within the dose range 5.10^4–2.10^6 r, $\mu(x)$ may be expressed approximately as

$$\mu(x)=\mu(0)e^{-\beta x} \tag{3}$$

where x is the depth in mm.

This effect is most pronounced with the low energy radiation employed because of the rapid fall in the "depth color curve," even within the range of glass thicknesses used (0.15–0.21 mm.).

Two slips of different thicknesses exposed to the same dose exhibit identical "depth color curves," but the average

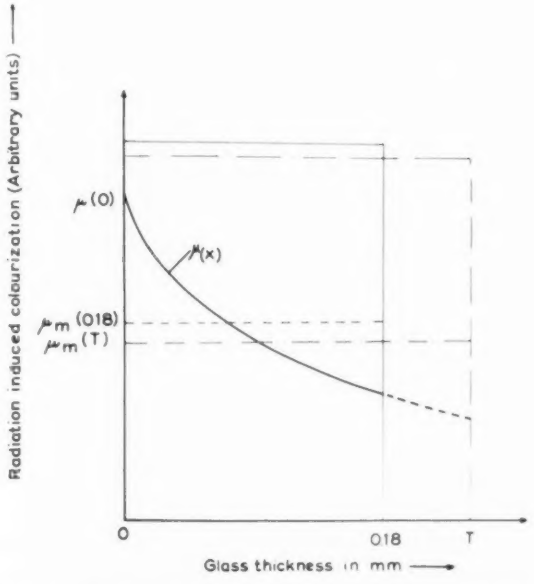

FIG. 2. The depth color curve through glasses of different thicknesses. The average colorization is indicated.

colorization μ_m (0.18) and μ_m (T) defined by formula (1) will be different (Fig. 2), and consequently also the measured doses. A correction term (k) which permits reference of all readings to the standard thickness of 0.18 mm. is therefore introduced.

The average colorization in the glass was assumed to be the total colorization homogeneously distributed through the glass, and may be expressed as

$$\mu_m(T)=\frac{1}{T}\int_0^T \mu(x)dx. \tag{4}$$

Consequently the correction term (k) is

$$k=\frac{\mu_m(0.18)}{\mu_m(T)}=\frac{T}{0.18}\cdot\frac{\int_0^{0.18}\mu(x)dx}{\int_0^T \mu(x)dx}. \tag{5}$$

Combining equation (3) and (5) the correction term (k) becomes

$$k=\frac{2.32T}{1-e^{-3T}} \tag{6}$$

which is presented graphically in Fig. 3.

Because of the nonlinear relationship between the dose and the colorization as pre-

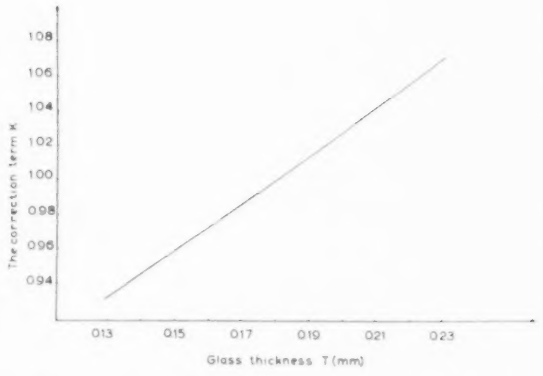

Fig. 3. The correction term (k) as a function of glass thickness.

sented by equation (2), the "depth color curve" and consequently (k) is dose dependent. The correction term, however, as expressed by equation (6) is correct at 3.10^5 r, but for practical purposes satisfactory results are obtained within the dose range 5.10^4 r to 2.10^6 r.

THE ENERGY DEPENDENCE OF THE DOSIMETER

The response of a glass dosimeter may be expected to change with the roentgen-ray spectrum. Investigations of this problem could not be found in the available literature. The glass dosimeter efficiency was therefore examined with varying external filtration of the 35 kev. roentgen irradiation. Studies carried out in this manner will, of course, cover a rather limited energy region, from half-value layers of 0.1 mm. Al up to about 1.3 mm. Al, corresponding roughly to an effective roentgen-ray energy-range of 10 kev.–25 kev.

A possible experimental procedure is to study the depth dose distribution in glass coverslips, in order to compare the glass dosimeter readings with a standard dosimeter system for increasing glass filtration.

Irradiations were performed through a glass stack the thickness of which was increased by successive additions of single coverslips. Dose determinations were effectuated using both radiation induced colorization as well as a high rate standard ionization chamber, constructed by Dr. N. H. Moxnes.[1] This set-up excludes backscatter which was expected to give a negligible contribution in the energy range with the field size (0.6 cm.^{2}) used here.[3,4] A check

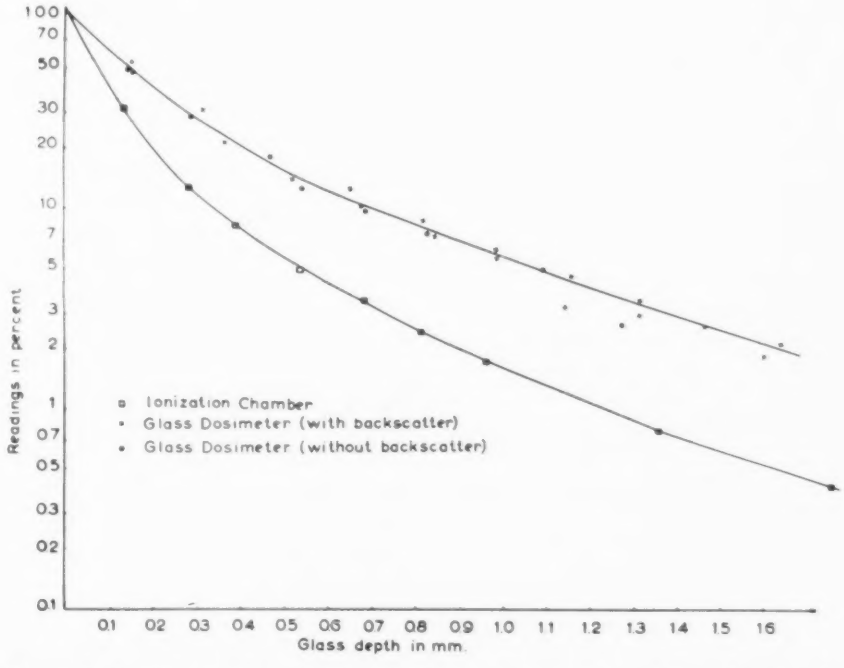

Fig. 4. Ionization chamber and glass dosimeter data, as a function of glass depth.

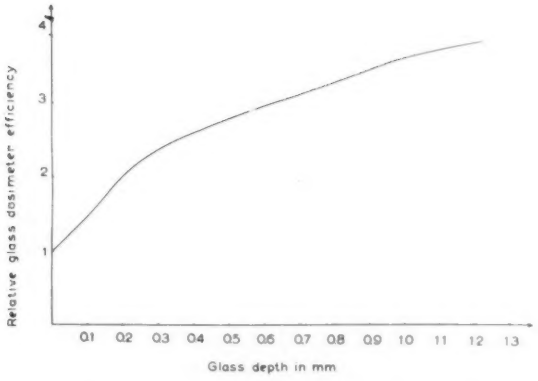

FIG. 5. The response of the glass dosimeter relative to a standard ionization chamber, as a function of increasing glass filtration.

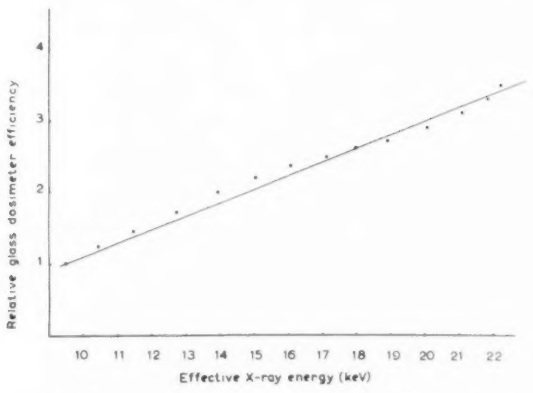

FIG. 6. The response of the glass dosimeter relative to a standard ionization chamber, as a function of the effective roentgen-ray energy.

was performed by irradiating a stack of coverslips of about 2 mm. thickness. Measurements of individual coverslips proved to coincide with the results obtained with the former method.

The observed data, corrected for inverse square law, and plotted on semilogarithmic paper, are shown in Figure 4.

In mathematical terms these curves may be represented by a function $D(x)$, where:

$$D(x) = \Sigma D_{0i} e^{-\mu_i x} \qquad (7)$$

D_{0i} is the contribution to the total surface dose of the monoenergetic line having absorption coefficient μ_i, and x is the depth below the glass surface, expressed in millimeters.[7]

For the present purpose, the depth dose distribution in the region $0 < x < 1.3$ mm. was under consideration, and in this interval, consideration of only two monoenergetic lines was necessary to give values sufficiently close to the experimental results obtained. The curve based on the ionization chamber data can then be expressed as

$$D(x) = 20e^{-2.5x} + 80e^{-11.7x} \qquad (8)$$

whereas the glass dosimeter data are given by

$$D(x) = 35e^{-1.8x} + 65e^{-7.2x} \qquad (9)$$

Dividing equation (9) by (8), the response of the glass dosimeter relative to the ionization chamber was obtained as a function of the depth. The results are shown in Figure 5.

The 35 kev. roentgen-ray spectrum may be characterized by its half-value layer in aluminum, and this in turn, gives the effective wavelength or energy.[10] The half-value layer of the radiation transmitted has thus been measured at various glass depths and the energy dependence of the glass dosimeter, relative to a specially calibrated ionization chamber, is presented in Figure 6. As shown in the figure, the efficiency of the glass dosimeter increases almost linearly with the energy in the range 10 to 23 kev.

In conclusion, the radiation induced colorization was utilized for a dosimeter which gave satisfactory measurements of doses from 10^4 to 10^7 r for the glass type and energy range specified. The dosimeter has small dimensions, is inexpensive, and is easy to use. The dosimeter should be suitable for use in studies of radiation effects on some unicellular organisms where large doses generally are necessary.

An extension of the energy region covered might prove interesting both from a practical and a theoretic point of view. Special attention should be paid to the position of the efficiency peak on the energy scale for glasses of different chemical composition.

SUMMARY

An investigation of a glass dosimeter for soft roentgen rays has been carried out. The

dose was determined from the radiation induced colorization in soda-silicate glass coverslips. Special efforts have been made to make the results independent of glass thickness. The energy dependence of the dosimeter system has been studied for 35 kev. roentgen rays with half-value layers ranging from 0.1 to 1.3 mm. Al.

Thanks are due to Chance Brothers Ltd., Birmingham, England, for information on the chemical composition of the glass coverslips used. Special thanks are due to Dr. Per Oftedal for the helpful discussion of this paper.

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RECOVERY OF ROENTGEN IRRADIATED *ESCHERICHIA COLI**

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RECOVERY of irradiated microorganisms under various environmental conditions has been observed by a number of investigators (Cook,[2] Latarjet,[4] Schrek,[9] Kelner,[3] Newcombe,[6] Stapleton *et al.*,[10] and Pratt *et al.*,[7]). The effects of low temperature storage on irradiated bacteria, however, have not been sufficiently explored to obtain general agreement.

In order to clarify some of the many variables which might be involved in the mechanism of low temperature recovery, the experiments described by Pratt, Moos and Eden[7] were repeated under identical conditions. The additional use of solid media for growth of parent cultures also allowed us to make comparisons with the results previously obtained by liquid (horse meat infusion) media. Our main aim in this investigation was to determine whether or not variation in the nutrient medium (liquid or solid) contributed to changes in the recovery pattern following roentgen irradiation.

MATERIALS AND METHODS

The organism was a strain of *Escherichia coli*, strain B, obtained from A. W. Pratt of the National Institutes of Health, Washington, D. C.

The strain was maintained prior to the start of the experiments by repeated biweekly transplantations in fresh veal infusion broth (Difco) and incubated at 36° C. The cultures (after 12 passages) were kept in the refrigerator at 2–4° C. and were again transplanted (four passages) to standard nutrient agar (Difco). At the time of the last culture transfer, organisms were deposited by standard platinum loop onto agar slant (horse meat infusion agar) or into broth (horse meat infusion broth) and incubated twenty-four hours at 36° C. After incubation, both broth and agar cultures were stored in the dark at 4° ± 1° C. and are from now on referred to as the "parent cultures." They were never allowed to warm up above 4° C. and were kept in an ice bath at all times when outside the refrigerator.

These parent cultures from either broth or agar slant kept at 4° C. were the source of organisms for all irradiation and control experiments. They were transferred by loop from either source into liquid medium (horse meat infusion broth, 10 ml.) and again incubated for twenty-four hours at 36° C., then centrifuged 3 times (20 min./cycle), and washed with double distilled water between each centrifugation cycle. Following the last centrifugation, the pellets were resuspended in 20 ml. distilled water by vigorous shaking with glass beads, and 0.1 ml. of each suspension added to 0.9 ml. of nutrient broth (Difco) in tubes intended for irradiation. The control (unirradiated) tubes received identical treatment. In the first series of experiments, studies were done with cultures from liquid media, and in the second series, cultures from agar slants were used.

At no time was the temperature of the samples (irradiated and controls) increased in either experiment beyond 4° C., even during the time of exposure to the radiation.

After irradiation, 9.0 ml. of sterile nutrient broth (Difco) was added to the tubes and they were held at 4° C.; these tubes from now on are designated "stock solutions." One milliliter of culture was removed daily or every second day up to the twelfth day from these "stock solutions"

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and then diluted $1:10^4$ with nutrient broth. One tenth of a milliliter of the final broth dilution ($1:10^4$) was seeded onto 5 standard nutrient agar plate surfaces in replicate, spread uniformly and incubated twenty-four hours at 36° C. Macrocolonies observed at this time were counted immediately and each point in the data presented represents the average of the 5 replicate counts.

The source of radiation was a Picker roentgen ray generator, 220 kv. peak with no filter except approximately 2 mm. of aluminum from the monitor ionization chamber. The distance from the target to the center of the test tube was 28.5 cm. The dose rate at this position was determined to be 131 r/min. The total dose usually applied to the samples amounted to 4,000 r.

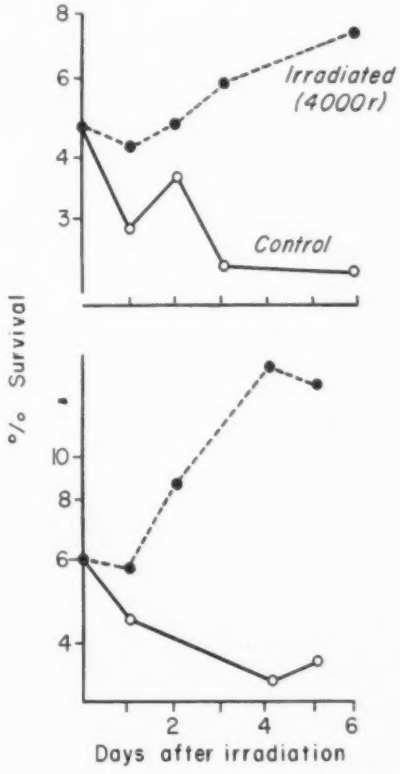


Fig. 1. Variation in the upper and lower graphs showing percentage survival of *E. coli* (strain B) originating from liquid (horse meat infusion broth) parent culture following irradiation and plated at intervals after roentgen-ray exposure.

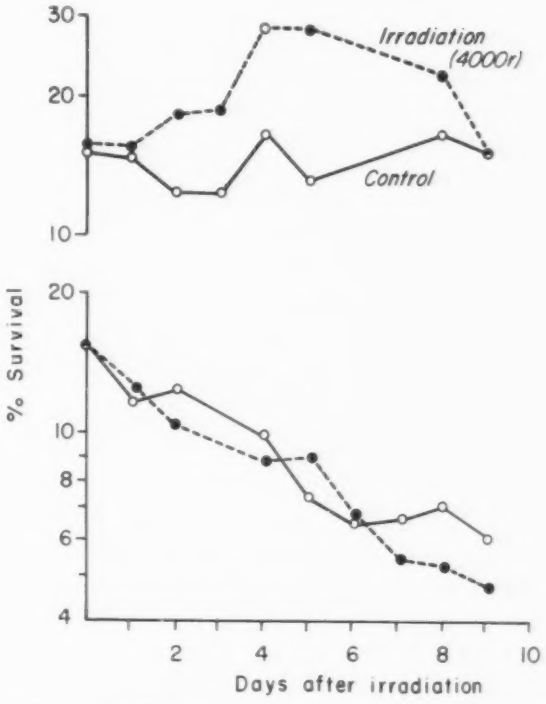


Fig. 2. Variation in the upper and lower graphs showing percentage survival of *E. coli* originating from solid (agar) parent culture demonstrating the infrequent and irregular recovery following irradiation and plated at intervals after roentgen-ray exposure.

Two series of experiments were carried out with samples from the liquid parent culture, and three series from the solid slant parent culture. Each series was tested at 5 to 16 various times as to the effect of aging on recovery. Recovery itself was followed over a period from five to twelve days.

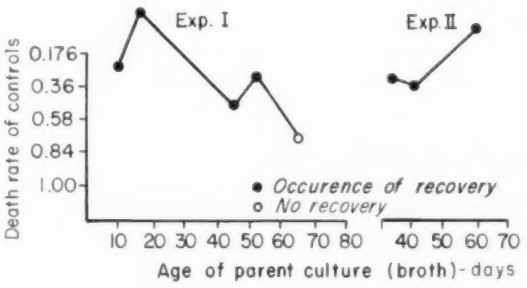
RESULTS

After exposure to roentgen irradiation, considerable recovery of *Escherichia coli*, strain B, cells was observed in almost every experiment when these cells came from a broth parent culture. This recovery effect was quite significant and very well defined when compared to control (unirradiated) cultures. Samples from a series of observations demonstrating recovery are shown in Figure 1.

The above experiments demonstrating recovery are in sharp contrast to the re-

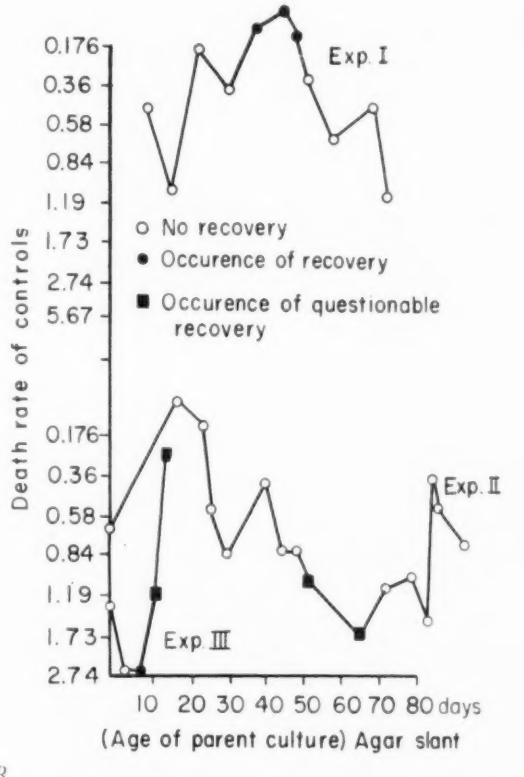
sults obtained in the experiments done with parent culture transplants from the solid media (agar slant) where only infrequent and irregular recovery was observed (Fig. 2).

The age of the parent cultures at the time of these experiments did not appear to be of critical nature since statistical analysis of the death rates of control *E. coli* cultures in either liquid or solid series indicated random variation. The instances where recovery was observed in irradiated cultures do not seem to follow a definite pattern if one attempts to correlate recovery with death rate of controls. Recovery of irradiated *E. coli* occurred when the death rate of the controls was either high or low, and at no specific age intervals of the parent cultures. These findings are illustrated in Figure 3*A* and Figure 3*B*. The age of the parent cultures can therefore be considered, to a certain extent, as less important to recovery than the phase of the medium in which the parent culture was maintained.



A

FIG. 3. (*A*) Death rate of *E. coli* control cultures respective to age of parent broth culture. Instances when recovery of irradiated cells occurred respective to age of parent broth culture are indicated by solid circles.



B

FIG. 3. (*B*) Death rate of *E. coli* control cultures respective to age of parent agar culture. Instances when recovery of irradiated cells occurred respective to age of parent agar culture are indicated by solid circles or squares.

DISCUSSION

In contrast to the work of Bretz and Hartsell,[1] who assume that dead cells at -9° C. furnish storage protection for *E. coli* survivors, we did not observe any recovery in the controls (unirradiated) at 4° C.

Microscopic examinations of older broth cultures (one to five weeks) stored at 4° C. exhibited organisms of considerable length, very much like those described by Maxwell and Nickel[5] and Whiteside-Carlson *et al.*[11] Such chain-like arrangements were observed only rarely in new broth cultures or in those from agar slants. It appears to us that these filamentous forms of *E. coli* are associated with the recovery effect. Whether this characteristic is contributory or just sequential has not been evaluated, and further experimentation is necessary to establish more evidence to clarify this mechanism. It may also be of interest to investigate whether recovery is associated with the long forms which have been observed in other organisms, *e.g.*, *Diplococcus pneumoniae* (Rochford and Mandle[8]).

SUMMARY

The recovery effect of *Escherichia coli*, strain B, following 4,000 r roentgen irradiation and kept at 4° C. has been shown to be greater in liquid than in solid media.

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THE EFFECT OF DIFFERENT DOSES OF ROENTGEN RAYS ON ADULT AXOLOTL (SIREDON MEXICANUM)*

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IN OUR previous paper,[3] the histopathology of roentgen death of young axolotl was described. The age factor is very important for radiosensitivity of animals and therefore we can expect reactions to irradiation of adult animals different from those of young animals. Investigation of the effect of roentgen rays on adult animals can give us new materials for understanding the biologic effect of irradiation. The sensitivity of adult axolotls to total body irradiation was not known, and the purpose of the present study is to investigate the effects of total roentgen irradiation on these animals.

The main problem considered in this paper is the comparison of the effects of various doses; therefore, the material is organized according to the doses that were used.

MATERIALS AND METHODS

In this investigation, 80 adult axolotls four to five years old were used. Ten animals served as untreated controls. Seventy animals were irradiated with the following doses: 500 r, 1,000 r, 3,000 r, 6,000 r, and 12,000 r. In each of the first three groups were 12 animals; 24 animals were irradiated with 6,000 r and 10 with 12,000 r.

Irradiations were made under the following conditions: 200 kv., 25 ma., 0.5 mm. Cu filter; target object distance, 25 cm.; intensity, 208 r/min. Irradiation of the last group of animals with 12,000 r was made under the following conditions: 250 kv., 30 ma., target object distance, 25 cm.; half-value layer, 0.95 mm. Cu; intensity, 685 r/min.

During irradiation, the animals were not anesthetized but restrained by means of gauze bandages. The animals were covered with a thin layer of moist cotton held in place by a bandage and then held in a coiled position by an additional bandage. As a result, it was possible to put a large animal in a small cardboard cottage cheese container. This container had a diameter on the bottom of 98 mm. and in the middle portion, about 114 mm. Accordingly, a target distance of 25 cm. could be used; therefore, the exposure time was considerably reduced. The container was filled with sufficient water to cover the animal almost completely.

In dead or dying animals, various tissues and organs were fixed for microscopic investigation. Fixations were made in a saturated solution of bichloride of mercury with 5 per cent acetic acid, in Zenker formol, and in acetic mercuric chloride formalin (Stieve). The material was embedded in paraffin with 5 per cent beeswax, and sectioned at 8 μ. The sections were stained with Ehrlich's hematoxylin and eosin. Photomicrographs were made at different magnifications (×37, ×160, ×450 and ×510).

RESULTS

The appearance and behavior of the irradiated animals during the first two to three weeks after irradiation with 6,000 r were usually normal. Only the animals in premortal state were distinguishable from the controls. They moved very slowly and somtimes turned on their backs. In some cases, it was possible to see new, light spots on the skin, and the gills were obviously reduced (Fig. 1, *A* and *B*). Such changes could be observed as early as the second week after irradiation with 12,000 r. Dissection of dead or dying animals

* This work was supported by grant from the National Cancer Institute, the United States Public Health Service.

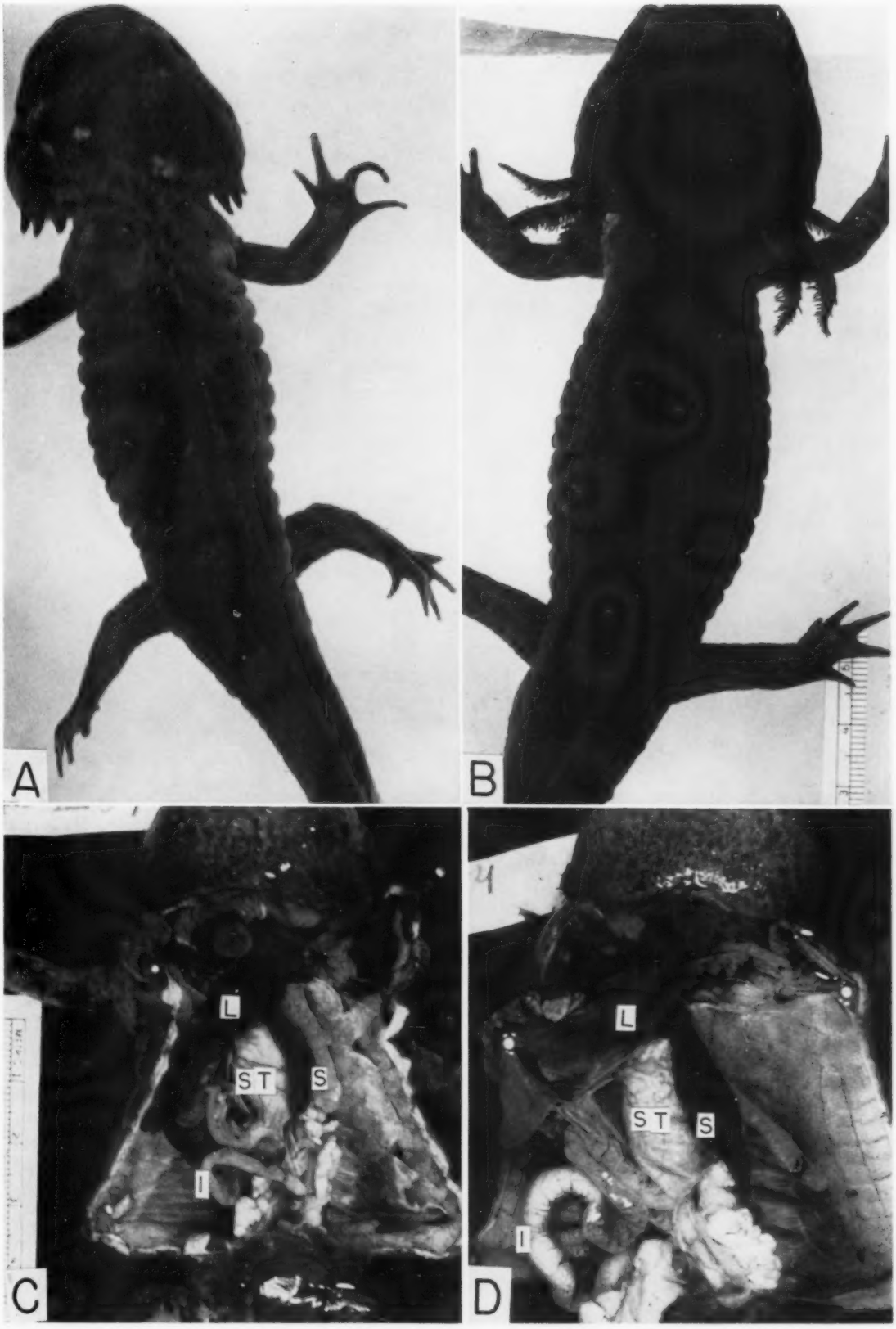


FIG. 1. *Irradiated and Control Animals.* (*A*) Axolotl (No. 11) 39 days after irradiation with 6,000 r. (*B*) Control axolotl of the same age. (*C*) Abdominal viscera of axolotl (No. 11) 39 days after irradiation with 6,000 r. (*D*) Abdominal viscera of control axolotl shown in *B*. I, small intestine; L, liver; S, spleen; ST, stomach.

showed that the spleen was greatly reduced in almost all cases, regardless of dose (Fig. 1, *C* and *D*). After irradiation with lower doses, premortal changes occurred much later.

1. THE SMALLEST DOSE—500 r

The dose of 500 r is very close to the tolerated dose for animals of such age. Animals of this group had a mean survival time of 196 days and exhibited a great variety of reactions. Some animals died comparatively early after irradiation (40–100 days after treatment), but about one-third of the animals of this group recovered completely; some were still alive more than two years after irradiation. The appearance of these animals was completely normal.

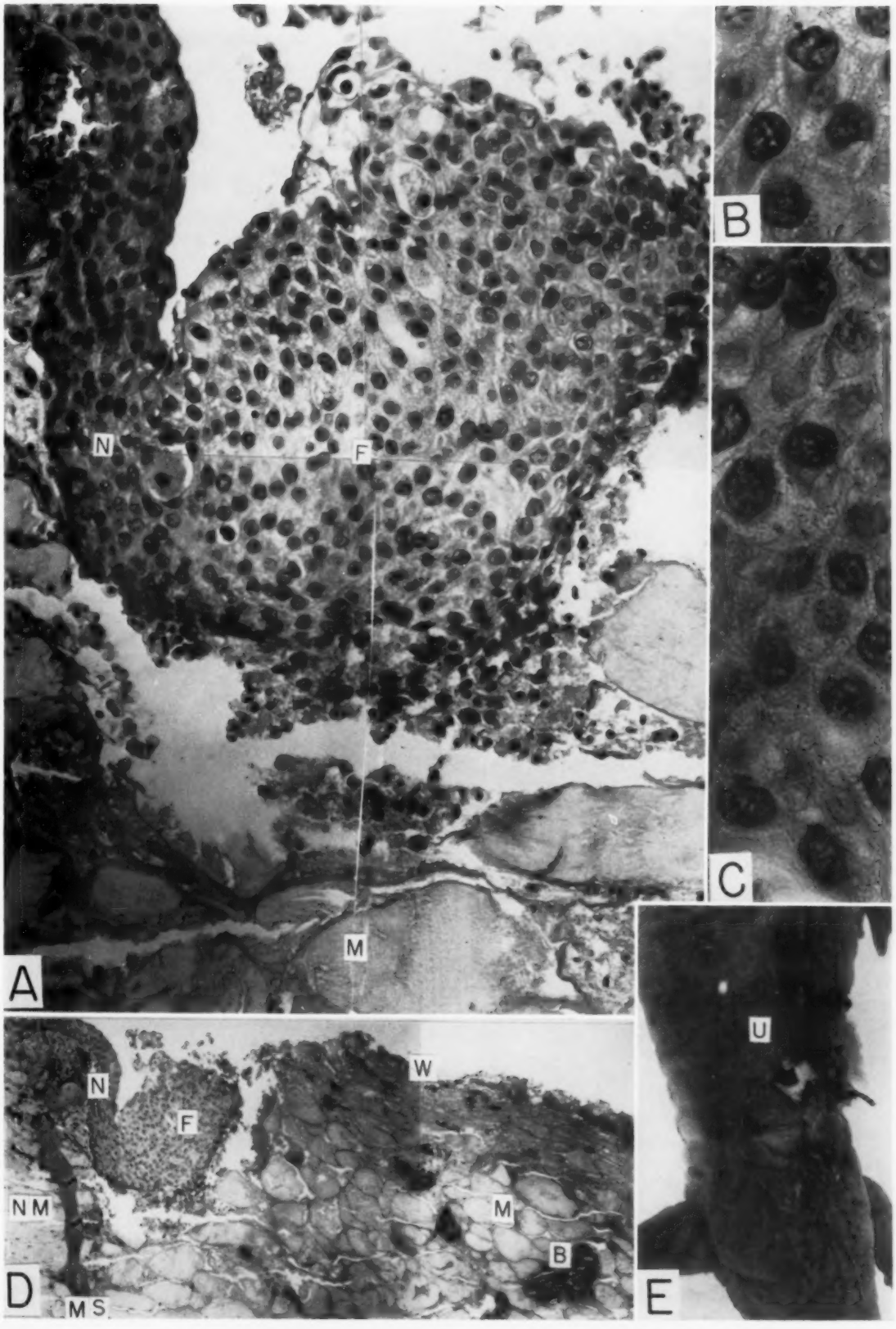
Results of Microscopic Examination. The damage of the skin is important for evaluation of roentgen injury in general. The appearance of the skin epithelium was normal in 100 per cent of cases after roentgen irradiation with this dose. Subepithelial tissues (connective tissue and muscles) were also normal in appearance. In spite of this, we have reason to think that some type of invisible changes occurred in the epithelium or in the subepithelial tissues. Only this hypothesis can explain the fact that late radiation necrosis of the skin was observed in some animals (22 per cent) of this group, 135–145 days after treatment. In one animal, this necrotic ulceration of the skin was extensive (Fig. 2 *E*) and the animal died. In another animal, a small necrotic ulcer was observed 145 days after irradiation. This ulcer later disappeared completely, and the animal was still alive more than two years after irradiation.

The histologic investigations of the first animal (No. 53) showed the following: epithelium of the skin near the ulcer was normal in appearance (Fig. 2 *D* and 3 *C*). In the boundary zone there was a very thick epithelial formation (Fig. 2 *A*) which consisted of slightly abnormal cells (Fig. 2, *B* and *C*). The nuclei of these cells were larger than normal. It is possible that this abnormality was connected with degeneration of subepithelial tissues. The outside surface of the ulcer itself had no epithelium (Fig. 2 *D*). The tissues of the ulcer consisted of damaged muscles, connective tissue and blood vessels. This injury was of considerable depth (Fig. 2 *D*).

Damage of the muscles was extensive and observed in 33 per cent of all animals irradiated with this dose, even in some animals that had no ulcer. This injury was demonstrated by the destruction and disappearance of nuclei and also by great changes in the fibers. The cross striation and longitudinal fibers in the ulcer region disappeared, and the muscle bundles were transformed into a completely homogeneous piece of tissue (Fig. 2, *A* and *D*; Fig. 3, *D* and *E*). It is necessary to note that this injury of the muscle fibers is very similar to that which was described after local irradiation with 7,500 r of the tails of tadpoles of Pelobates fuscus.[14] Similar observations were made by Lewis[10] in striated muscle of male albino rabbits after irradiation with very large doses: 72,000 r, 36,000 r, 24,000 r and 12,000 r. Soon after irradiation, complete coagulation necrosis of irradiated muscle was noted. The muscle was transformed into amorphous masses with loss of cross striations. No abnormalities could be observed after administration of 6,000 r.

Damage to the connective tissue was obvious in some myosepta (dense connec-

FIG. 2. *Roentgen Ulcers.* Sections through the skin ulcer (U) of axolotl (No. 53) 135 days after irradiation with 500 r and appearance of the animal with ulcer (*E*). (*A*) Section through the caudal portion of the ulcer. Photomicrograph ×160. (*B* and *C*) Epithelial cells by larger magnification from thick epithelial formation. Photomicrograph ×510. (*D*) Section through the caudal and central portions of the ulcer. Photomicrograph ×37. B, blood vessels, abnormally wide; F, very thick epithelial formation; M, abnormal degenerating muscles; MS, myoseptum; N, epithelium of normal thickness; NM, muscles normal in appearance; W, surface of the ulcer without epithelium.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tive tissue between muscle segments) which were abnormally thick in the central portion of the ulcer. Damage to the blood vessels was evident from the expansion of some vessels in the ulcer region (Fig. 2 *D*) and from the great subepithelial hemorrhage which was observed in some places near the ulcer (Fig. 3 *A*). It is noteworthy that the skin epithelium in some places above this hemorrhage was abnormally thin and obviously close to rupture (Fig. 3, *A* and *B*). Probably this is the explanation of the disappearance of epithelium from the surface of the ulcer.

The epithelium of the small intestine was normal in 100 per cent of the cases. Damage of connective tissue was noted only in the ulcer region. The cartilage also was normal in all of the cases. Damage of the spleen was important in determining the lethal effect of roentgen rays. This damage was noted in 55 per cent of all animals and, in 33 per cent, it was serious. For example, in the spleen of an animal dying 43 days after irradiation with 500 r, the number of lymphocytes was greatly reduced and large numbers of erythrocytes were present. Perhaps, if these animals had continued to live after irradiation, some undamaged cells of the spleen would have been able to aid in the recovery of this organ. This is a possible explanation of the fact that the spleen of most animals, fixed a long time after irradiation with this dose, was normal in appearance. For example, the animal (No. 51) killed more than one year after irradiation with 500 r had a spleen with a normal number of lymphocytes (Fig. 4 *C*). However, in the spleen of some animals it was possible to see unusually large lymphocytes between the normal lymphocytes. These abnormal cells can be considered as evidence of some irradiation effect. In some animals, light damage of the spleen (reduction of the number of lymphocytes) was noted even a long time after irradiation. Perhaps in these cases recovery was incomplete.

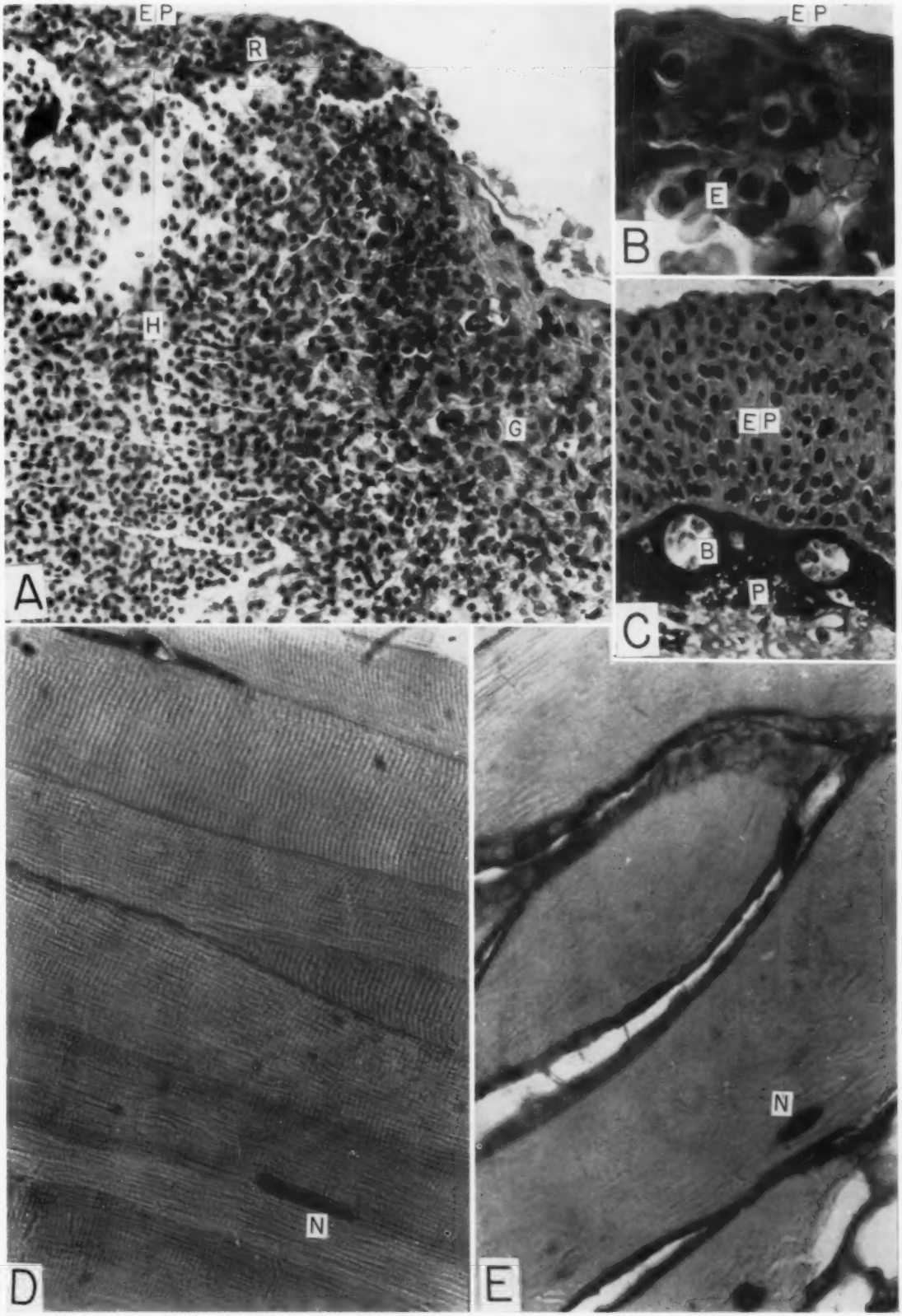
Slight damage of the liver was observed in 44 per cent of all animals irradiated with this dose. Some of the liver cell nuclei were enlarged.

It was noted in our previous paper[3] that the peripheral portion of the liver must be considered separately because this portion of the organ, in axolotl, has a structure and function completely different from all of the other portions of the liver. This is typically lymphoid tissue, which is very similar to the lymph nodes and the spleen. Like other lymphoid organs, this portion of the liver is very sensitive to roentgen irradiation. In 44 per cent of all animals irradiated with this dose, this layer was completely absent. In 1 animal (Fig. 5 *B*), this layer was abnormal, consisting of giant degenerating cells (compare with Fig. 5 *A*), and in other cases it was normal. It is necessary to take into consideration that normal peripheral portions of the liver were found only in animals fixed a long time after irradiation when obvious recovery had occurred. For example, this was observed in the liver of animal No. 51, killed 398 days after irradiation with 500 r.

2. THE MEDIUM DOSES—1,000 and 3,000 r

The mean survival time of animals after irradiation with 1,000 r was 55 days and after irradiation with 3,000 r, 44 days.

FIG. 3. *Roentgen Ulcer and Damage of the Muscles.* (*A–C*) Section through the skin near the ulcer of axolotl (No. 53) 135 days after irradiation with 500 r. (*A*) Subepithelial hemorrhage near the caudal portion of the ulcer. Photomicrograph ×160. EP, very thin abnormal epithelium; G, thick abnormal epithelium with abnormally large nuclei; H, hemorrhage under epithelium; R, region of abnormally thin epithelium shown at large magnification (×510) in *B*; EP, epithelium; E, erythrocytes. (*C*) Epithelium normal in appearance near the ulcer. Photomicrograph ×160. B, blood vessel; EP, epithelium; P, pigment layer. (*D*) Section through the muscles in the head of control animal. Photomicrograph ×510. (*E*) Section through the muscles in the head of axolotl (No. 57) 109 days after irradiation with 500 r. Photomicrograph ×510. N, nucleus.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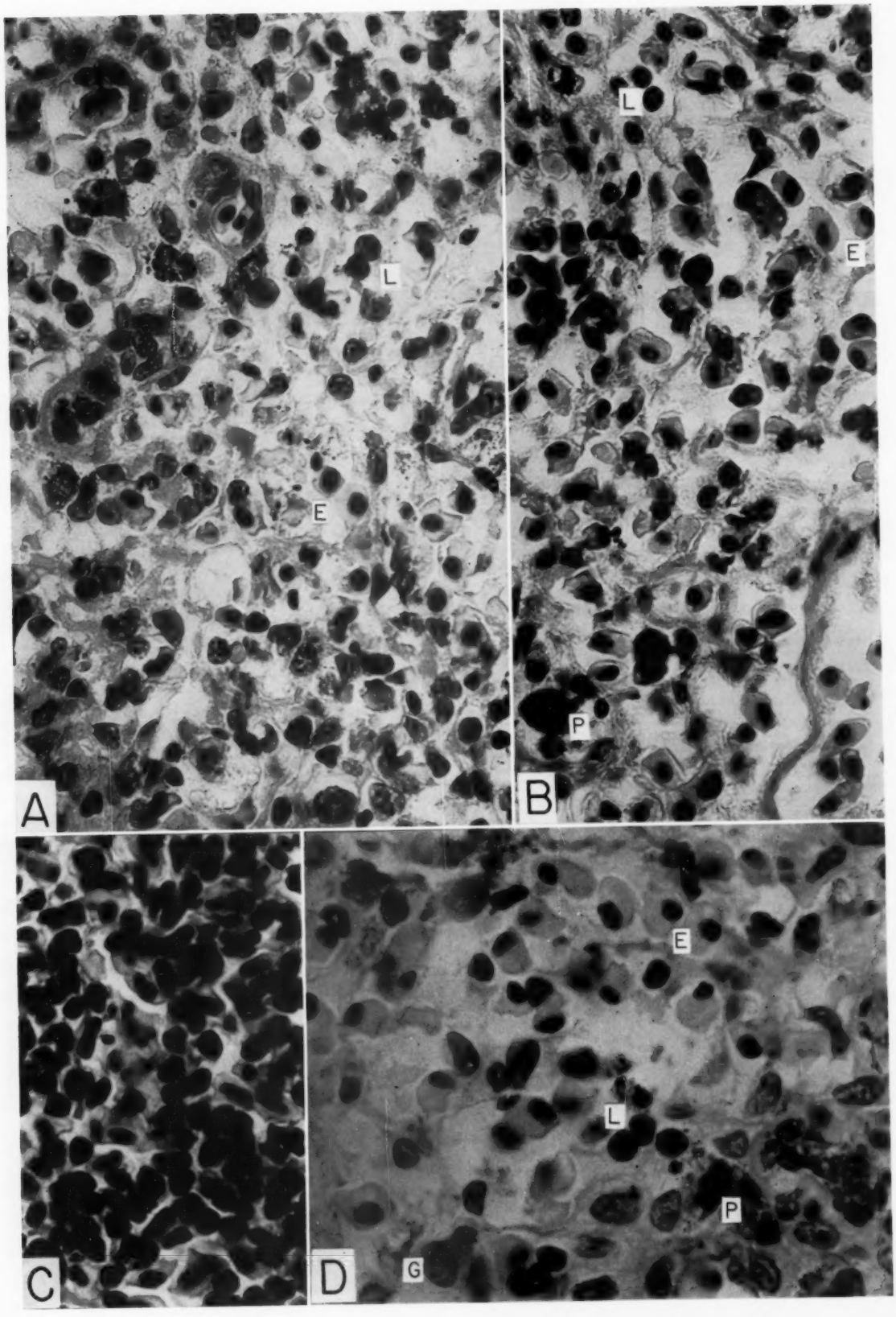
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Results of Microscopic Examination. Damage of the epithelium was noted in 71 per cent of all animals irradiated with these doses, but only in some cases did the formation of giant cell epithelium occur. In other cases, the epithelium was only slightly abnormal; the cells were larger than normal and had atypical appearance. In general, it is necessary to take into consideration that, in spite of total body irradiation, the complete and uniform damage to tissues or organs was not observed in many cases. Some portions of skin epithelium were clearly damaged; in other portions this damage was not obvious, and some portions were completely normal in appearance.

Damage of the muscles was observed in only 25 per cent of all animals after irradiation with these doses. In all of these cases, the nuclei were slightly larger than normal, while the muscle fibers were normal.

Damage of connective tissue was observed in only 22 per cent of the cases. In one case of damage, some of the fibroblasts were abnormally large. The cartilage was normal in 100 per cent of the cases. Slight damage of the epithelial cells of the small intestine (enlargement of the nuclei) was noted in only 30 per cent of all cases.

Obvious damage of the spleen was observed in all of the cases. It is necessary to emphasize that, in 75 per cent of the cases, this damage was of serious consequence. In most cases, the number of lymphocytes was greatly reduced, and an unusually large number of erythrocytes was accumulated (Fig. 4 *D*). Some of the irradiated spleens were almost completely empty and only a few cells were present.

In 72 per cent of irradiated livers, slight damage was noted. The liver cells were larger than normal. In 90 per cent of the livers, the peripheral (lymphatic) portion was completely absent. In the others, an abnormal layer with giant cells was seen.

3. THE HIGH DOSE—6,000 r

The mean survival time of animals after irradiation with this dose was 28 days.

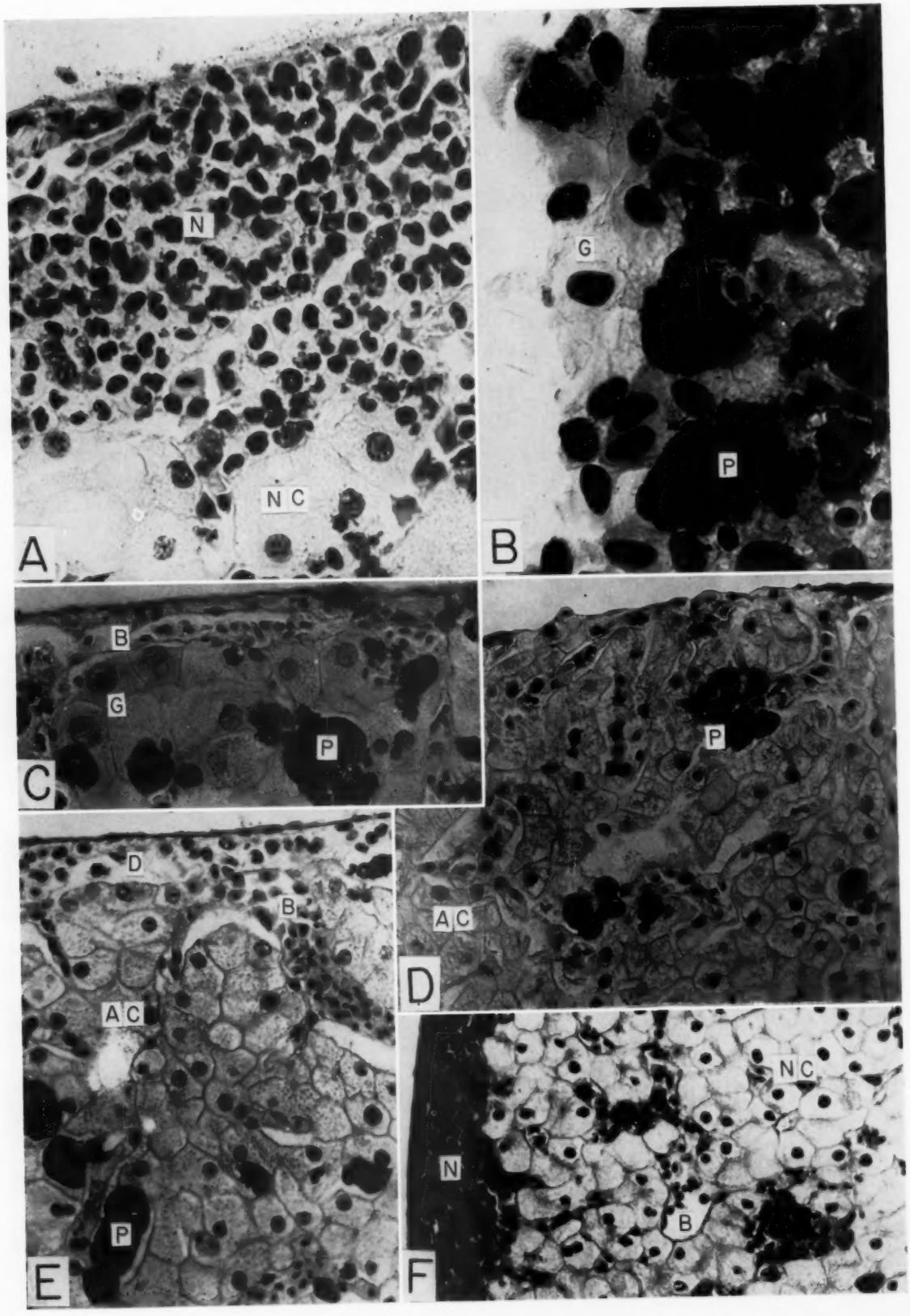
Results of Microscopic Examination. After irradiation with this dose, the damage to skin epithelium, spleen and liver was more obvious than after irradiation with smaller doses. Slight to moderate damage to skin epithelium was noted in 87 per cent of all cases. Considerable or extensive damage of the spleen occurred in 87 per cent of irradiated animals (Fig. 4, *A* and *B*) and in 13 per cent it was moderate. In the liver, slight damage of cells was noted in 81 per cent of all cases. The peripheral portion of the liver was absent in 100 per cent of the cases. The cartilage was normal in all cases after irradiation. In the intestinal epithelium, connective tissue and muscles, slight damage was noted in 25 per cent of all cases.

4. THE HIGHEST DOSE—12,000 r

The mean survival time of animals after irradiation with 12,000 r was 11 days. We can consider this as acute roentgen death. It is noteworthy that the minimal length of life for animals after irradiation with this dose was 7 days and the maximal was 18 days. It was apparent that all animals died before the primary acute reactions to irradiation could be observed (according to our previous observations, this reaction can be observed between 20 and 60 days following treatment[2,4]). From this standpoint, the histologic changes in tissues after irradiation with this dose were the most interesting.

Results of Microscopic Examination. An obvious damage of skin epithelium after

FIG. 4. *Damage of the Spleen.* (*A*) Section through the spleen of axolotl (No. 11) 39 days after irradiation with 6,000 r. Photomicrograph ×450. (*B*) Section through the spleen of axolotl (No. 15) 17 days after irradiation with 6,000 r. Photomicrograph ×450. (*C*) Section through the spleen of axolotl (No. 51) 398 days after irradiation with 500 r. Normal appearance. Photomicrograph ×450. (*D*) Section through the spleen of axolotl (No. 38) 51 days after irradiation with 3,000 r. Photomicrograph ×450. E, erythrocytes; G, abnormally large (giant) lymphocyte; L, lymphocytes, normal in size; P, pigment cell.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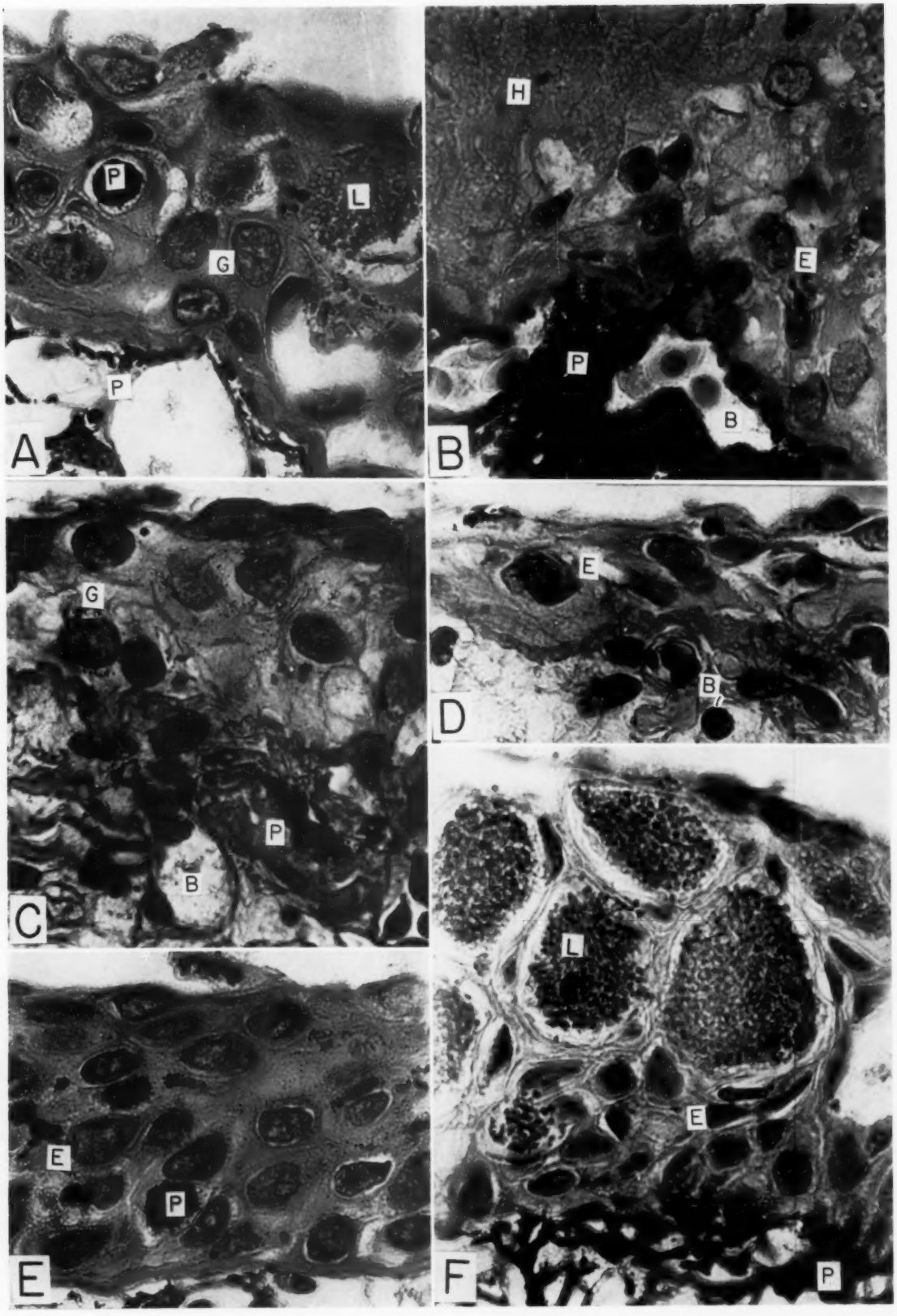
irradiation with this dose was observed in 87 per cent of all animals. Mostly, this damage was great and very variable. The common sign was disappearance of Leydig's cells, which are very numerous in normal epithelium of adult animals (Fig. 6 *F*). This cell can be noted only occasionally in irradiated epithelium (Fig. 6 *A*). The epithelial cells usually transformed into large or giant cells with very large nuclei (Fig. 6, *A*, *C* and *E*). The reduction in the number of epithelial cells was typical (Fig. 6, *A* and *C*). In some places, irradiated epithelium had only one layer of cells (Fig. 6 *D*) instead of the normal six to twelve layers (Fig. 6 *F*). It is noteworthy that the appearance of the skin epithelium in various places of the same animal was very different. In some places it was normal, in others it was damaged. The damage, too, differed greatly. For an illustration of this, it is interesting to compare Figure 6, *B* and *E*. Both photomicrographs were made from various places of epithelium of the same animal which died 13 days after irradiation. On Figure 6 *E* we see atypical epithelium with large nuclei and without Leydig's cells. On Figure 6 *B* the size of nuclei is normal, but part of the epithelium transformed into homogeneous tissue without nuclei and cell border. We have never before observed such damage of epithelium. It is necessary to emphasize that ulcers of the skin were noted in 50 per cent of investigated animals after irradiation with this dose. Microscopic examination of these ulcers showed that near the ulcer some portions of epithelium were normal in appearance, but between these portions it was possible to see epithelium which had transformed into homogeneous tissue as described above. We came to the conclusion that such degenerated epithelium was an initial phase of formation of roentgen ulcer. Obvious damage of subepithelial tissues in this group was not observed and, therefore, it is very possible that ulceration was a result of damage of the epithelium itself.

Abnormalities of epithelium of the gills were noted in all of the investigated animals. In some instances there was only atypical configuration of the epithelium and presence of large nuclei. But in other cases obvious degeneration of the gill epithelium occurred (Fig. 7, *D* and *E*).

Damage of the spleen was noted in 87 per cent of all cases. But in all cases, this damage could be evaluated as moderate only. Serious damage was not observed. On the contrary, the damage of the liver in some animals was more extensive than after irradiation with smaller doses. In 12 per cent of all cases the nuclei of cells in some portion of the liver had a giant size (compare Fig. 5, *C* and *F*). In 37 per cent of all animals the liver cell nuclei were not as large as in the first described cases, but still definitely enlarged (Fig. 5, *D* and *E* compared with Fig. 5 *F*). In 51 per cent of the cases, damage was only slight; the nuclei of the liver cells were slightly enlarged.

The peripheral portion of the liver was absent in 75 per cent of all animals (Fig. 5, *C* and *D*). But in 25 per cent of all animals, definite remnants of peripheral lymphatic

FIG. 5. *Damage of the Liver.* (*A*) Section through the liver of control animal. Photomicrograph ×450. N, normal peripheral (lymphatic) portion of the liver; NC, normal liver cells. (*B*) Section through the liver of axolotl (No. 52) 72 days after irradiation with 500 r. Photomicrograph ×450. G, degenerating lymphatic cells of giant size; P, pigment cells. (*C*) Section through liver of axolotl (No. 2) 13 days after irradiation with 12,000 r. Photomicrograph ×160. B. blood vessel; G, liver cells with giant nuclei; P, pigment cells. (*D*) Section through the liver of axolotl (No. 9) 13 days after irradiation with 12,000 r. Photomicrograph ×160. AC, abnormal liver cells; P, pigment cell. (*E*) Section through the liver of axolotl (No. 8) 8 days after irradiation with 12,000 r. Photomicrograph ×160, AC, abnormally large nuclei of liver cells; B, blood vessel; D, degenerated cells of peripheral portion of the liver. (*F*) Section through the liver of control animal. Photomicrograph ×160. B, blood vessel; N, normal peripheral portion of the liver; NC, normal liver cells.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layer were noted. The great difference between such peripheral portion and normal one is obvious (Fig. 5, *E* and *F*).

The epithelium of the small intestine showed some interesting peculiarities which were not discovered after irradiation with smaller doses. In 100 per cent of all animals, the secreting cells of intestinal epithelium (goblet cells) were damaged. In some cases this damage was very great as shown in Figure 7 *B*. Mucus produced by these cells was not excreted normally and therefore accumulated in more or less great amounts. The appearance of such epithelium was clearly distinguishable from the normal intestinal epithelium of control animals (Fig. 7, *A* and *B*). In other cases, the accumulation of mucus was not as great, but obviously abnormal (Fig. 7, *C*). The nuclei of these epithelial cells were also abnormal. They were larger than those of the intestinal epithelium of control animals (Fig. 7, *A* and *B*).

The nuclei of epithelial cells in the cloaca were also larger than in control animals.

The muscles and cartilage in all investigated animals of this group were normal. In connective tissue some fibroblasts were abnormally large in 25 per cent of all cases.

DISCUSSION

The comparison of all data concerning tissues and organs damaged after total body irradiation with various doses is shown in Table 1. The surprising conclusion is that some similarity can be noted in the results of irradiation with the smallest (500 r) and the largest (12,000 r) doses. The similarity extends to the skin, spleen, and the peripheral portion of the liver. Only after irradiation with these two doses did ulceration of the skin occur and the damage to the spleen was less than after irradiation with medium doses; the peripheral portion of the liver did not disappear completely in all cases and, therefore, abnormally large lymphatic cells in this zone were observed. The possible explanation of this similarity is the following: The dose of 500 r is too weak to provoke the quick death of irradiated animals; therefore, in most cases they live long enough for observation of late reaction (late radiation necrosis) which was not observed in other cases. This reaction can be compared with late gangrenous dermatitis which was described in a man several years after irradiation.[12] Furth and co-workers,[6] described various types of late radiation effects in mice after total body irradiation. For example, fatal degeneration of the kidney was observed eighteen months after irradiation with 500 r. Lyman and co-workers[11] found that some dogs developed signs of diffuse damage to the functions of the cortex and subcortex five months after local irradiation of the head with 500 r. According to Warren,[16] five years were required after the atomic bomb explosion for the development of radiation cataracts in the survivors. The dose of 500 r is also too weak for complete destruction of lymphatic tissue of the spleen and the peripheral portion of the liver. Consequently, if animals did not die soon after irradiation, the complete recovery of these organs was observed.

The dose of 12,000 r is large enough to provoke ulceration of the skin in 50 per

FIG. 6. *Damage of the Skin.* (*A*) Section through the skin of axolotl (No. 3) 14 days after irradiation with 12,000 r. Photomicrograph ×510. G, abnormally large nuclei of epithelial cells; L, Leydig's cell; P, pigment cell. (*B*) Section through skin of axolotl (No. 2) 13 days after irradiation with 12,000 r. Photomicrograph ×510. B, blood vessel; E, epithelial cells; H, degenerated epithelium transformed into homogeneous tissue; P, pigment cell. (*C*) Section through skin of axolotl (No. 9) 13 days after irradiation with 12,000 r. Photomicrograph ×510. B, blood vessel; G, abnormally large nuclei of epithelial cells; P, pigment cell. (D) Section through skin of axolotl (No. 6) 7 days after irradiation with 12,000 r. Photomicrograph ×510. B, blood vessel; E, epithelial cells. (*E*) Section through skin of axolotl (No. 2) 13 days after irradiation with 12,000 r. Photomicrograph ×510. E, epithelial cells; P, pigment cell. (*F*) Section through skin of control animal. Photomicrograph ×510. E, epithelial cells; L, Leydig's cell; P, pigment cell.

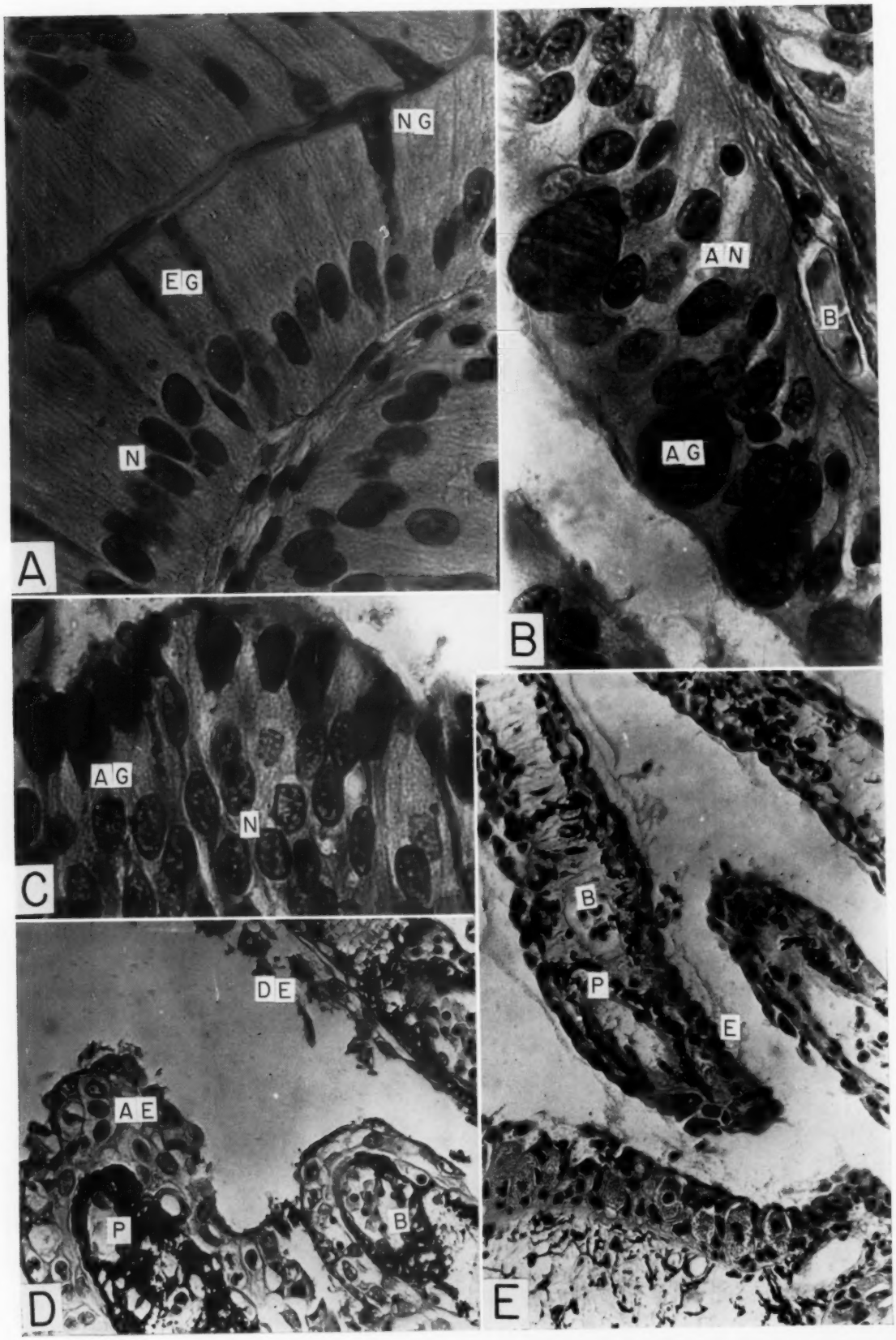
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TABLE I

COMPARISON OF EFFECTS OF DIFFERENT DOSES OF ROENTGEN RAYS ON TISSUES AND ORGANS OF ADULT AXOLOTL

Doses	Epithelium of the Skin	Ulcers	Epithelium of the Gills	Epithelium of the Small Intestine	Loose Connective Tissue	Cartilage	Muscles	Spleen	Liver	Peripheral Portion of the Liver
500 r	normal*	22%	normal	normal	normal	normal	33% serious damage	55% serious and moderate damage	44% slight damage	44% absent 11% abnormal 45% normal
1,000 r and 3,000 r	71% slight damage	absent†	not investigated	30% slight damage	22% slight damage	normal	25% slight damage	100% very serious, serious and moderate damage	72% slight damage	90% absent 10% abnormal
6,000 r	87% slight and moderate damage	absent†	not investigated	25% slight damage	25% slight damage	normal	25% slight damage	100% very serious, serious and moderate damage	81% slight damage	100% absent
12,000 r	87% serious and moderate damage	50%	100% moderate and serious damage	100% moderate damage	25% slight damage	normal	normal	87% moderate damage	100% slight, moderate and serious damage	75% absent 25% abnormal

* Normal in appearance in 100% of cases.
† Absent in 100% of cases.

cent of all irradiated animals. It is clear that animals after irradiation with this dose died chiefly from damage of skin epithelium and gills and possibly also from damage of small intestine and liver, but not from damage of lymphatic organs such as spleen and peripheral portions of the liver. On the contrary, damage of lymphatic organs (spleen and peripheral portion of the liver) was possibly the main cause of death of animals irradiated with other doses (including the 500 r dose). Therefore, the main conclusion from the comparison of effect of different doses of roentgen rays is that the mechanism of roentgen death differs with various doses of irradiation. Accordingly, Prosser[13] distinguished six types of roentgen death in higher animals: (1) Immediate death at very high doses with general cellular destruction. (2) Initial shock-like death seen within 48 hours after irradiation. There is vasodilatation, erythema, and general gastrointestinal dysfunction. (3) Early death at high doses after 4–6 days.

FIG. 7. *Damage of the Epithelium of the Intestine and the Gills.* (*A*) Section through the intestinal epithelium of control animal. Photomicrograph ×510. EG, empty goblet cells; NG, normal goblet cell with mucigen; N, nuclei. (*B*) Section through the intestinal epithelium of axolotl (No. 4) 10 days after irradiation with 12,000 r. Photomicrograph ×510. AN, abnormally large nuclei; AG, abnormal goblet cell; B, blood vessel. (*C*) Section through the intestinal epithelium of axolotl (No. 8) 9 days after irradiation with 12,000 r. Photomicrograph ×510. AG, abnormal goblet cells; N, nuclei. (*D*) Section through the gills of axolotl (No. 10) 16 days after irradiation with 12,000 r. Photomicrograph ×160. B, blood vessel; AE, abnormally large gill epithelium; DE, degenerating gill epithelium; P, pigment cells. (E) Section through the gills of control animal. Photomicrograph ×160. B, blood vessel; E, gill epithelium; P, pigment cells.

There is evidence of dehydration, gastrointestinal damage and leukopenia. (4) Acute death occurring 0–21 days after treatment. There is evidence of extensive tissue breakdown, leukopenia and slight anemia. (5) Subacute death due to slow pathologic changes. There is evidence of anemia, liver degeneration, bone marrow lesions. (6) Chronic death. Tumor development is an important cause of death, but other types of chronic irradiation injury were also observed.

Similarity of ulceration of the skin after irradiation with 500 r and 12,000 r probably is only superficial. With the low dose, the epithelium of the skin was normal and damage of it was secondary to damage of subepithelial tissues such as muscles, connective tissue and blood vessels. After irradiation with 12,000 r, the subepithelial tissues were not damaged and ulceration was the result of injury of the epithelium itself.

The theory of primary acute radiation reaction followed by a secondary stable state (Brunst,[2] Brunst and co-workers[4]) must be revised and adapted to these new observations. First, it is necessary to point out that the formerly described reactions were established as a result of observations on local radiation effects. Of course, a great difference exists in radiation effects between local and total irradiation because all phenomena connected with the boundary zone between irradiated and nonirradiated portions of the body (macrophages, inflammation and "zone of stimulation") disappear in case of total irradiation.

The local radiation effects were investigated by treatment of tails, limbs, and lumbosacral regions of axolotls, so that the spleen or peripheral portion of the liver were not included in the irradiated area. These organs are highly radiosensitive and, of course, can alter the general picture of reaction of the organism to irradiation. The statement which must be changed is the time when the primary acute reaction sets in. According to our previous observations, this reaction occurs between 20 and 60 days after treatment, but his is not the case in all instances. In general, this is correct for the skin epithelium by local and total irradiation. However, we do have some exceptions. For example, in our previous paper[3] it was mentioned that normal epithelium was found in some cases as early as 39 days after treatment. In studying the mouse limb more than one year after local irradiation with 3,000 r,[5] the skin areolar and adipose tissues were noted to be essentially normal in appearance, but the cartilage and bone, although normal in some places, showed the greatest disturbance in other regions. In the material described in this paper, the damage to the spleen was observed most frequently between 20 and 60 days after irradiation, but also 16, 65, 72 and more than 100 days after treatment. It is noteworthy that some animals died 16 days after irradiation with 3,000 r, and 8 days after irradiation with 6,000 r. After irradiation with 12,000 r, all animals died between 7 and 18 days following treatment. Thus one may conclude that the primary acute reaction is observed in most cases between 20 and 60 days after treatment if the dose of irradiation is not too large. However, after total body irradiation with very large doses, such as 12,000 r, the animals die very quickly and the primary acute reaction sets in almost immediately after treatment. Histologic changes described in this paper after irradiation with 12,000 r are very similar to those seen after irradiation with smaller doses. In connection with this, it is interesting to note the observation of Gerstner, Powell and Richey[8] as regards the influence of very large doses (above 150,000 r) on isolated striated frog muscle. These authors found an immediate and complete abolishment of muscle function associated with morphologic changes. Severe histologic alterations in rabbit muscles were seen 24 hours following irradiation.[7] According to Brace, Andrews and Thompson,[1] a characteristic tetanic state developed in guinea pigs after irradiation with 10,000–25,000 r. This was noted less than an hour after termination

of the exposure and persisted until the animals were moribund. If the dose is high enough the death may occur "under the beam," *i.e.*, during the exposure. Henshaw[9] gave doses of 25,000 r to 50,000 r of roentgen rays at 250 r per minute to mice, rabbits and guinea pigs. The animals died during exposure or a few hours later. Rugh, Levy and Sapadin[15] gave a dose of 110,000 r to hamsters. In animals which died during the exposure, severe morphologic changes were described.

SUMMARY

1. In this investigation, 80 adult axolotls four to five years old were used. Ten served as untreated controls. Seventy animals were irradiated with doses of 500 r, 1,000 r, 3,000 r, 6,000 r and 12,000 r.

2. The mean survival time of animals after irradiation was as follows: for 12,000 r—11 days; for 6,000 r—28 days; for 3,000 r—44 days; for 1,000 r—55 days. The dose of 500 r is very close to the tolerated dose for animals of such age. The mean survival time for this group was 196 days. About one-third of the animals of this group recovered completely and some of them lived more than two years after irradiation.

3. Comparative study of the effects of different doses of roentgen rays on the tissues and organs of adult axolotl (Table 1) showed the following: (a) Some similarity was noted in the results of irradiation with the smallest (500 r) and largest (12,000 r) doses. The similarity extended to the skin, spleen and the peripheral portion of the liver. Only after irradiation with these two doses did ulceration of the skin occur and the damage to the spleen was less than after irradiation with medium doses; the peripheral portion of the liver did not disappear completely and abnormal lymphatic cells on this zone were observed. (b) The animals after irradiation with 12,000 r died chiefly from damage of the skin epithelium and gills and possibly also from damage of the small intestine and liver, but not from damage of the lymphatic organs such as spleen and the peripheral portion of the liver. On the contrary, damage of the lymphatic organs (spleen and peripheral portion of liver) possibly was the main cause of death of animals irradiated with other doses (including the 500 r dose). (c) The main conclusion from the comparison of these effects of different doses of roentgen rays on adult axolotl is that the mechanism of roentgen death differs with various doses of irradiation.

4. A primary acute reaction can be observed after total body irradiation, but with great modification. In most cases, it can be noted between 20 and 60 days after irradiation, if the dose of roentgen rays is not too large. However, after total body irradiation with very large doses, this reaction can set in almost immediately.

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Fifty-ninth Annual Meeting: **Shoreham Hotel, Washington, D. C., September 30–October 3, 1958.**

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Forty-first Annual Meeting: The Homestead, Hot Springs, Virginia, April 6–8, 1959.

EDITORIALS

FIFTY-NINTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Fifty-ninth Annual Meeting of the American Roentgen Ray Society will be held at the Shoreham Hotel, Washington, D. C., Tuesday, Wednesday, Thursday, and Friday, September 30–October 3, 1958. Previous meetings there have been so successful that the Society is returning with much enthusiasm. To those attending is promised a warm reception and interesting program by the Committee on Local Arrangements, headed by Dr. Edgar M. McPeak and by the Ladies' Committee, chairmanned by Mrs. Edgar M. McPeak. The hospitality of the hotel and the interest in our Nation's capital are other reasons for large registrations when the Society convenes in Washington. It is hoped that all of those who plan to attend will write immediately to the Shoreham Hotel, Washington 8, D. C., for reservations.

The Commercial Exhibits will be set up in the West Lobby Foyer, Main Ball Room, Foyer, Foyer A and Bird Cage Walk. Dr. James C. Cook, Manager of the Annual Meeting, reports an unusual demand for space and a large participation is expected. The Commercial Exhibits will be open from 8:30 A.M. to 5:00 P.M., as arranged by "Cliff" Sherratt.

Doctor Cesare Gianturco, Chairman of the Scientific Exhibits Committee, and the other members of this Committee, Doctors Richard H. Marshak and Ted F. Leigh, have succeeded in obtaining numerous excellent exhibits with wide appeal and marked teaching value. The Committee plans to keep the Exhibits open from 8:00 A.M. to 8:00 P.M. so that all interested will have ample time to study the material.

The Annual Golf Tournament for all members and guests will be held once more at the Manor Country Club, Monday, September 29. Doctor Ralph M. Caulk, Chairman of the Local Golf Committee and a member of the Club, has made arrangements for us to have the usual good time when visiting this fine club. Luncheon, golf and dinner will be enjoyed by all attending and entertainment following the dinner will include a showing of movies of previous tournaments by Arthur Fuchs and his associates from Eastman Kodak. At the conclusion of the dinner, the Willis F. Manges Trophy for the Eighteen Hole Medal Handicap and the Exhibitors' Trophy for the Eighteen Hole Gross Score will be awarded, along with other prizes. Buses will transport members from the hotel to the club and will return following the end of the evening activities.

Registration begins at 2:00 P.M. on Sunday, September 28, and will be continued each day from 8:00 A.M. to 5:00 P.M. The Registration Desk will be in the Foyer leading to the Commercial Exhibits.

The Scientific Sessions will be held in the Terrace Banquet Room, Tuesday through Friday, beginning at 8:30 A.M. and ending at 12:00 P.M. The meeting will be called to order on Tuesday morning, September 30 by President Wendell G. Scott, after which Doctor Wilbur Bailey, Chairman of the Executive Council, will assist Doctor Scott in the installation of the President-Elect, Barton R. Young.

The Preliminary Program will be found elsewhere in this issue of the JOURNAL. The Program Committee, which started work on this year's meeting at the conclusion of last year's session and has continued its efforts

in order to insure the best possible program, decided that it would be worthwhile to have at least two feature lectures on subjects with wide appeal to radiologists in the Scientific Sessions, and selected Doctor Robert R. Newell to open the program with a dissertation entitled "Common-Sense Evaluation of Radiation Exposure in Clinical Radiology." Doctor Newell is eminently qualified to give this type of address and everyone attending can look forward to an outstanding presentation. Additional papers related to this problem will follow. One of these is "Radiation and the Community. A New Role for the Radiologist" by Doctor George Tievsky. Another is "Incidence of Neoplasia after Irradiation of the Thymic Region" by Doctors H. B. Latourette and F. J. Hodges. Following these presentations is a group of papers on cerebral arteriography and on diseases affecting the skull and brain.

The Caldwell Lecture will be given Tuesday evening, September 30, in the Terrace Banquet Room at 8:15 P.M. by Dr. Leo G. Rigler, President of the Radiological Society of North America. His title is "Functional Roentgen Diagnosis—Anatomical Image—Physiological Interpretation." Doctor Rigler's outstanding ability as a lecturer and his reputation are reasons to anticipate a capacity audience. He will be introduced by a long-time associate, student and personal friend, Doctor Harold O. Peterson, who succeeded him as Professor of Radiology at the University of Minnesota.

The first portion of the Wednesday morning session will be used for the presentation of a group of five very fine papers dealing with pulmonary diagnosis. These will be followed by papers on coronary and aortic arteriography. The last half of the morning session will be devoted to the presentation of four papers dealing with diagnosis of disease of the alimentary tract.

Thursday morning's program will be opened by a feature address by Doctor Ernest E. Aegerter, Professor of Pathology at Temple University, who will speak on the subject of "What the Radiologist Should Know about the Pathology of Bone Tumors." Throughout most of his professional life, Doctor Aegerter has studied bone tumors and has a new concept that should be challenging not only to radiologists and pathologists but to everyone interested in the study of bone tumors. Following this lecture there will be several papers on bone, joint and renal disease.

For Friday morning, a number of papers pertaining to radiation therapy are scheduled and three of these will deal with a study of radiation from as yet rarely used radioactive isotopes of cesium, yttrium and sulphur. The meeting will be concluded with a paper on "The Joy and Cost of Office Radiology" by Doctor John L. McClenahan, of Philadelphia. This promises to be of unusual interest and therefore will merit consideration by all attending the meeting.

The business meetings of the Society will be held at 4:30 P.M. in the Terrace Room on Tuesday and in the Park Room on Thursday.

Doctor Harold O. Peterson, Director of the Instructional Courses, has made arrangements for more than 64 courses with unusual interest and variety. The latest developments and ideas in diagnostic and therapeutic fields will be presented by a fine Faculty. A number of new instructors have consented to participate and a goodly number of previous instructors will be on hand to assist. The Instructional Courses will be held each afternoon, Tuesday through Friday, from 3:00 to 4:30 P.M. with the exception of Friday, when the courses will be held at 1:30 to 3:00 P.M. Members and guests will be required to fill out application blanks for the courses they desire, and will receive tickets in advance of the meeting. In the August issue of the JOURNAL details of the Instructional Courses will be published.

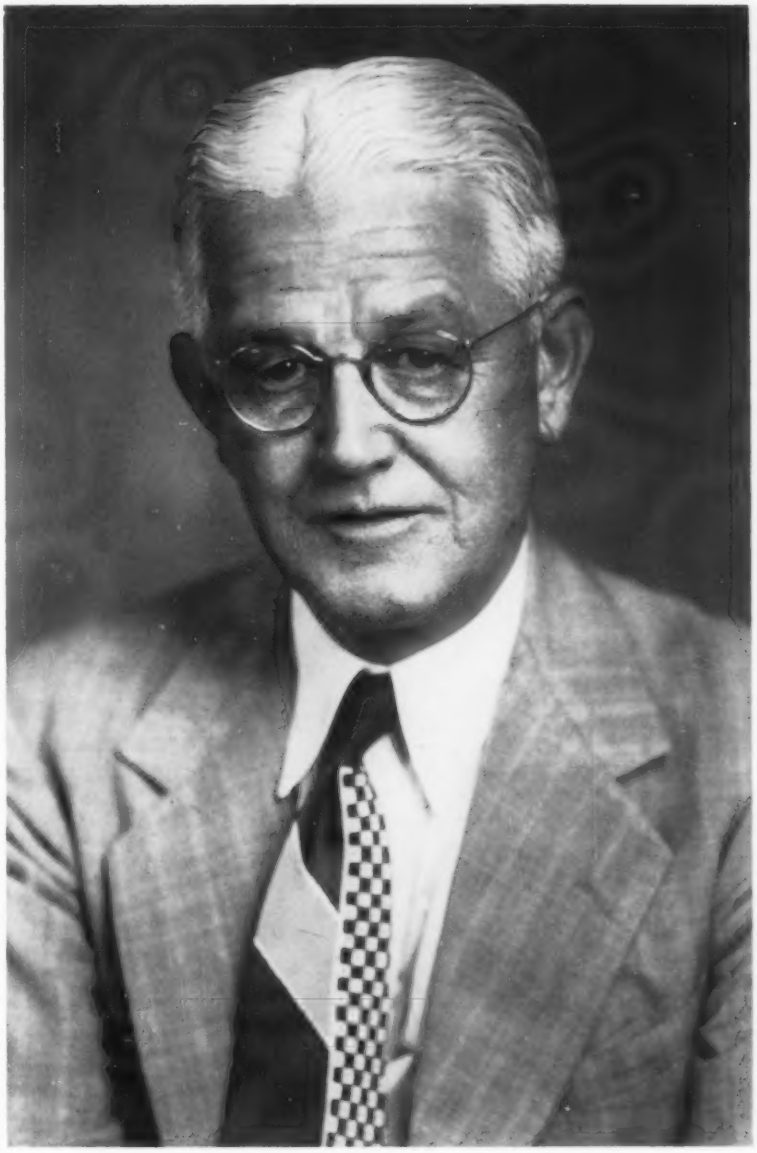
The meetings in Washington have always been popular with the ladies, largely due to the fine facilities and the excellent program arranged by the Ladies' Committee. Mrs. Edgar M. McPeak has arranged a theater party (Cinerama, "Search for

Paradise"), for 8:30 P.M., Monday, September 29 for the ladies and any members who wish to attend. Buses will be provided for transportation. On Tuesday, September 30, there will be a sightseeing tour to Annapolis, Maryland, by bus, leaving the Shoreham Hotel at 12:00 noon, with luncheon at Carvel Hall in Annapolis. A Fashion Show and luncheon will be held in the Park Room of the Shoreham, Wednesday, October 1 and on the following day, Thursday, October 2, there will be a sightseeing tour of Washington, which will include the White House and Wax Museum, with luncheon at the Water Gate Inn on the Potomac River. The highlight of the Meeting, the Annual Banquet, will be held Thursday evening preceded by a cocktail party at 6:30 P.M. at the Shoreham Hotel.

A cordial invitation is extended not only to the members of the Society, but to all radiologists, their wives and guests, to attend this annual meeting.

Barton R. Young, M.D.
President-Elect

Germantown Hospital
Philadelphia 44, Pennsylvania



EUGENE THOMAS LEDDY
1892-1958

EUGENE THOMAS LEDDY, for several years head of the Section of Therapeutic Radiology at the Mayo Clinic, died in Rochester, Minnesota on January 5, 1958. He had come to Rochester on October 1, 1925 as first assistant in Therapeutic Radiology; in 1929 he was appointed a consultant and advanced from instructor in Radiology in the Mayo Foundation, Graduate School, University of Minnesota, to Assistant Professor in 1933 and Associate Professor in 1937. He was certified as a specialist in Radiology by the American Board of Radiology in 1934. He became a senior consultant in Radiology in 1953 and retired from active practice in October, 1957.

Dr. Leddy was born in Taunton, Massa-

chusetts on September 27, 1892. He graduated from Harvard with a bachelor of arts degree in 1915 and that of doctor of medicine in 1919. Following an internship at Memorial Hospital in New York and at Carney Hospital in Boston, he became an assistant in 1920 at the Memorial Hospital and from 1922 to 1925 he was head of the Roentgen-Ray Department of the Philadelphia General Hospital. He practiced roentgenology in Providence, Rhode Island, before coming to the Mayo Clinic in 1925.

Dr. Leddy devoted his life to treatment of cancer and allied diseases and was an acknowledged leader in this field. He carried out much research on possible injuries due to radiation therapy and on safeguards against them. His teaching ability in training the fellowship men at the Mayo Clinic was outstanding. He made many contributions to medical literature and was conversant with many foreign languages, enabling him to keep abreast of the literature in his field. His ready Irish wit always made him a delightful companion.

He was a Fellow of the American College of Radiology, and a member of the American Medical Association, The American Radium Society, The American Roentgen Ray Society, the Radiological Society of North America, the Alumni Association of the Mayo Foundation, and the Society of the Sigma Xi.

Dr. Leddy was married to Miss Anna C. Regan on June 8, 1926. He is survived by Mrs. Leddy and two children, John Eugene Leddy and Anna Marie Leddy.

Robert E. Fricke, M.D.

CLARENCE ELTON HUFFORD
1893-1958

ON MARCH 25, 1958, while motoring in Florida to attend the meeting of The American Radium Society, Dr. C. E. Hufford was suddenly stricken and passed away. He had not been, to the knowledge of his close friends, in poor health, although he had had some cardiac difficulty of a moderate degree some two years previously.

At the time of his death Dr. Hufford was prominent in many activities, both locally and on a national level. He was Director of the Department of Radiology at St. Vincent's Hospital, Toledo, Ohio, where his constant attention to detail, his sincere feeling for each and every patient, and his sterling qualities as a physician endeared him to patients and staff.

Dr. Hufford gave much of himself to Radiology. His activities in the Radiological Society of North America, of which he was president in 1956, in the American College of Radiology, which he served as chancellor for a number of years, and in the Ohio Division of The American Cancer Society are but a few in which he was engaged.

Dr. Hufford graduated from Western Reserve Medical School in 1920 and his entire medical life was spent in Toledo.

After a period in the general practice of medicine, be became associated with the late Dr. John T. Murphy in Radiology in 1929 and carried on in that practice following Dr. Murphy's death.

Dr. Hufford is survived by his wife Crystal, his mother, Mrs. Amaretta Hufford, a daughter Virginia, and three grandchildren.

Clarence Hufford will long be remembered by his associates and friends as an understanding gentleman, a comforting physician, and a stellar radiologist.

F. C. CURTZWILER, M.D.

PRELIMINARY PROGRAM

FIFTY-NINTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

The Fifty-ninth Annual Meeting of the American Roentgen Ray Society on September 30 to October 3, 1958 will again be held at the Shoreham Hotel, Washington, D. C.

The Executive Council will meet on Sunday, September 28.

As last year, the Manor Country Club will be the site of the Annual Golf Tournament for the Willis F. Manges Trophy for the Eighteen Hole Medal Handicap and the Exhibitors' Trophy for the Eighteen Hole Gross Score. After the tournament, the two trophies and also other prizes will be awarded at the Annual Golf Dinner.

This year's Caldwell Lecture will be delivered on Tuesday evening, September 30 by Dr. Leo G. Rigler, President of the Radiological Society of North America, in the Terrace Banquet Room.

The Instruction Courses will be held each afternoon, Tuesday through Friday. A detailed plan of the courses will be published in the August issue of the JOURNAL.

President-Elect Barton R. Young and his Committee have arranged the Scientific Program of the Meeting, which is as follows:

Tuesday, September 30, 1958

Terrace Banquet Room

8:30 A.M. Call to Order, Fifty-ninth Annual Meeting, Wendell G. Scott, M.D., President. Address of Welcome: Major General James P. Cooney, Deputy Surgeon General, Department of the Army.

Installation of the President-Elect, Barton R. Young, M.D., Philadelphia, Pennsylvania, by President Wendell G. Scott, M.D., St. Louis, Mo., and Wilbur Bailey, M.D., Chairman of the Executive Council, Los Angeles, Calif.

Inaugural Address: President Barton R. Young, M.D. The American Roentgen Ray Society. Its Previous, Present-Day and Future Importance to Radiology and Medicine.

9:15 A.M.

Presiding: Barton R. Young, M.D.
Philadelphia, Pa.

1. Common-Sense Evaluation of Radiation Exposure in Clinical Radiology. Robert R. Newell, M.D., San Francisco, Calif.
2. Radiation and the Community: A New Role for the Radiologist. George Tievsky, M.D. (by invitation), Washington, D. C.
3. Incidence of Neoplasia after Irradiation of Thymic Region. Howard B. Latourette, M.D. (by invitation) and Fred J. Hodges, M.D., Ann Arbor, Mich.
4. The Angiographic Study of Carotid Insufficiency and Cerebral Ischemia. Marvin M. Keirns, M.D. (by invitation), and John E. Whiteleather, M.D., Memphis, Tenn.
5. Radiologic Aspects of Cerebral Angiography in Children. Juan M. Taveras, M.D., and Charles M. Poser, M.D. (by invitation), New York, N.Y.
6. The Incisura of the Cerebellar Tentorium. Herniation through the Tentorial Slit As Revealed by Vertebral Angiography. H. F. Plaut, M.D. (by invitation), Dayton, Ohio.
7. The Roentgenological Manifestations of Optic Nerve Glioma. Colin B. Holman, M.D. (by invitation), Rochester, Minn.
8. Roentgenological Features of Meningeal Fibroblastoma. Sidney P. Traub, M.D. (by invitation), Saskatoon, Saskatchewan, Canada.

Discussion of Papers 4 to 8 inclusive to be opened by D. L. McRae, M.D., Montreal, Quebec, Canada.

Tuesday Evening, September 30, 1958
Eight o'clock

THE CALDWELL LECTURE

Functional Roentgen Diagnosis—Anatomical Image—Physiological Interpretation
by
Leo G. Rigler, M.D.

Consultant in Radiology, Cedars of Lebanon Hospital
Visiting Professor of Radiology, University of California at Los Angeles
President, Radiological Society of North America
Introduction—
Harold O. Peterson, M.D.
Professor of Radiology
University of Minnesota
Minneapolis, Minnesota
Presentation of the Caldwell Medal to Dr. Rigler by President Young

Wednesday, October 1, 1958

8:30 A.M.

Presiding: Eugene P. Pendergrass, M.D.
Philadelphia, Pa.

9. Idiopathic Unilateral Radiolucent Lung. Lee S. Rosenberg, M.D. (by invitation), Harold N. Margolin, M.D. (by invitation), and Benjamin Felson, M.D. (by invitation), Cincinnati, Ohio.
10. Tomography in the Radiological Evaluation and Differentiation of Chronic Hypertrophic Emphysema and Asthma. Robert G. Fraser, M.D. (by invitation), and David V. Bates, M.D. (by invitation), Montreal, Quebec, Canada.
11. The Importance of Chest Roentgenography in the Diagnosis of Pulmonary Embolism. George N. Stein, M.D. (by invitation), Jen Ti Chen, M.D. (by invitation), Harold L. Israel, M.D. (by invitation), Arthur Finkelstein, M.D., and Franz Goldstein, M.D. (by invitation), Philadelphia, Pa.
12. Investigation of Effects of Opaque Media on the Lungs with Comparison of Barium Sulphate, Lipiodol and Dionosil. B. Skinner, M.D. (by invitation), G. Wortzman, M.D. (by invitation), J. R. Stuart, M.D. (by invitation), and J. S. Dunbar, M.D. (by invitation), Montreal, Quebec, Canada.
13. Positive Roentgenograms Plus Positive Sputum Smears Do Not Always Equal Pulmonary Tuberculosis. C. Allen Good, M.D., D. T. Carr, M.D. (by invitation), and L. A. Weed, M.D. (by invitation), Rochester, Minn.

Discussion of Papers 9 to 13 inclusive to be opened by Leo G. Rigler, M.D., Los Angeles, Calif.

14. Coronary Arteriography. J. Stauffer Lehman, M.D., Randal A. Boyer, M.D. (by invitation), and Fred S. Winter, M.D. (by invitation), Philadelphia, Pa.
15. Contrast Material Dissection of Aortic Wall in Direct Aortography. D. E. Boblitt, M.D. (by invitation), and Melvin M. Figley, M.D., Ann Arbor, Mich.
16. Roentgen Demonstration of Lesions Mimicking Malignancy of the Cardiac End of the Stomach. George T. Wohl, M.D., and Leah Shore, M.D. (by invitation), Philadelphia, Pa.
17. Importance of Emptying Film in Roentgen Examination of Upper Gastrointestinal Tract to Detect Unrecognized or Inconclusively Demonstrated Ulcer, Carcinoma and Other Disease. Jay W. MacMoran, M.D. (by invitation), Robert B. Funch, M.D. (by invitation), and Barton R. Young, M.D., Philadelphia, Pa.
18. Radiologic Aspects of Hydrostatic Reduction of Ileo-Colic Intussusception. Bertram R. Girdany, M.D. (by invitation), Lee W. Bass, M.D. (by invitation), William K. Sieber, M.D. (by invitation), and Geno DiBagno, M.D. (by invitation), Pittsburgh, Pa.
19. Roentgen Abnormalities in Disease of the Appendices Epiploicae. David Kirsh, M.D., and Rudolph E. Drosd, M.D. (by invitation), Miami, Fla.

Discussion of Papers 16 to 19 inclusive to be opened by Philip J. Hodes, M.D., Philadelphia, Pa.

Thursday, October 2, 1958

8:30 A.M.

Presiding: John P. Medelman, M.D.
White Bear Lake, Minn.

20. What Every Radiologist Should Know about Bone Tumors. Ernest E. Aegerter, M.D. (by invitation), Philadelphia, Pa.
21. Osteoid Osteoma. A Review of 80 Cases. Robert H. Freiberger, M.D. (by invitation), Milton Helpern, M.D. (by invitation), Bernard S. Loitman, M.D. (by invitation), and T. Campbell Thomp-

son, M.D. (by invitation), New York, N.Y.

22. Radiation Induced Dysplasia of Bone. Concepts in Bone Modeling. Philip Rubin M.D. (by invitation), Rochester, N. Y., Richard L. Swarm, M.D. (by invitation), J. Robert Andrews, M.D., and Hazel Gump (by invitation), Bethesda, Md.
23. Joint Infections following Steroid Therapy; Roentgen Manifestations. Roderick L. Tondreau, M.D., Philip J. Hodes, M.D., and Erwin R. Schmidt, Jr., M.D. (by invitation), Philadelphia, Pa.
24. Myelofibrosis: The Radiologic Findings in Twenty-Five Proven Cases. Ted F. Leigh, M.D., Charles C. Corley, Jr., M.D. (by invitation), Charles M. Huguley, Jr., M.D. (by invitation), and James V. Rogers, Jr., M.D., Emory University, Ga.
25. The Visual Physiology of Roentgen Diagnosis. A Technique for the Evaluation of Radiologic Methods. William J. Tuddenham, M.D. (by invitation), Philadelphia, Pa.
26. Idiopathic Adrenal Calcification in Infants and Children. J. Luther Jarvis, M.D. (by invitation), and William B. Seaman, M.D., New York, N.Y.
27. Roentgenographic Analysis of Renal Mass Lesions (Cysts and Tumors). Lawrence Reynolds, M.D., Harold Fulton, M.D. (by invitation), and James J. Snider, M.D., (by invitation), Detroit, Mich.
28. Radioisotope Renography. John A. Evans, M.D., New York, N.Y.
29. A Critical Evaluation of External Body Surface Counting in the Determination of Cardiac Output with Radioactive Isotopes. Barbara L. Carter, M.D. (by invitation), Sigurd E. Johnsen, M.D. (by invitation), R. Kenneth Loeffler, M.D. (by invitation), and Martha E. Southard, M.D. (by invitation), Philadelphia, Pa.

Friday, October 3, 1958

8:30 A.M.

Presiding: John E. Whiteleather, M.D.
Memphis, Tenn.

30. The Physical Aspects of a Cesium Teletherapy Source. Charles S. Simons, Ph.D. (by invitation), Isadore Lampe, M.D., and Howard B. Latourette, M.D. (by invitation), Ann Arbor, Mich.
31. The Significance of the Tumor Bed Reactions in the Radiation Treatment of Malignant Tumors. Halvor Vermund, M.D. (by invitation), Madison, Wisc.
32. The Combination of Oxygen and Optimum Fractionation in Radiation Therapy of Adenocarcinoma. Lucille A. DuSault, A.B. (by invitation), William R. Eyler, M.D. (by invitation), and Glen D. Dobben, M.D. (by invitation), Detroit, Mich.
33. The Radiotherapeutic Management of Certain Complications of Childhood Leukemia. G. J. D'Angio, M.D. (by invitation), Anna T. Mitus, M.D. (by invitation), and Audrey Evans (by invitation), Boston, Mass.
34. Hodgkin's Disease. Combined Roentgen and Nitrogen Mustard Therapy As Compared to Roentgen Therapy Alone. James C. Cook, M.D., and Kenneth L. Krabbenhoft, M.D., Detroit, Mich.
35. Results of Radiation Treatment of Carcinoma of Tonsil and Tonsillar Pillars Treated from 1932–1956. Norah duV. Tapley, M.D. (by invitation), Morton M. Kligerman, M.D., Robert A. Evans, M.D., and Harold Jacox, M.D., New York, N.Y.
36. Trans-Sphenoidal Destruction of the Hypophysis with Radioactive Yttrium. Robert D. Moseley, Jr., M.D. (by invitation), William M. S. Ironside, M.D. (by invitation), and Paul V. Harper, M.D. (by invitation), Chicago, Ill.
37. The Immediate and Late Radiation Effects of One Curie of S-35 Administered Internally to a Case of Chondrosarcoma. J. Robert Andrews, M.D., Richard L. Swarm, M.D. (by invitation), Lawrence Schlachter, M.D. (by invitation), Kirkland C. Brace, M.D. (by invitation), Delbert M. Bergenstal, M.D. (by invitation), Robert W. Swain, B.S. (by invitation), Philip Rubin, M.D. (by invitation), Hazel Gump (by invitation), and Sidney Siegel, M.S. (by invitation), Bethesda, Md.
38. Radiographic Visualization of Lymph

Nodes and Lymphatic Channels. Harry W. Fischer, M.D. (by invitation), and George R. Zimmerman, M.D. (by invitation), Iowa City, Iowa.

Discussion—Robert Robbins, M.D., Philadelphia, Pa.

39. The Joy and Cost of Office Radiology. John L. McClenahan, M.D., Philadelphia, Pa.

NATIONAL COMMITTEE ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

THE increased uses of radiation in both medicine and industry have resulted in a sharp increase in interest in the work of the National Committee on Radiation Protection. This committee was activated in 1928 with the concurrence of the various radiological societies in an effort to consolidate our national recommendations for presentation on an international basis. The Committee has been in effective operation since then, ever growing and enlarging its terms of reference as the needs arose; at present its recommendations are widely recognized and used in the medical, industrial, military, and related applications of radiation, and have become the principal legal support for the enforcement of radiation protection practices in the United States. Many of the recommendations have also been adopted in principle or *in toto* by the corresponding international groups dealing with radiation protection and measurements.

Sponsorship of the Committee encompasses many interests in the field including the major radiological societies, the American medical and dental associations, industrial and labor groups, and government agencies interested in atomic energy, public health, and military aspects. The Committee is operated by the National Bureau of Standards as a public service, and its

RECOMMENDATIONS OF THE NATIONAL COMMITTEE ON RADIATION PROTECTION AND MEASUREMENTS

NBS Handbook No.	Title	Date Issued	Price
42	Safe Handling of Radioactive Isotopes	Sept. 1949	$.20
48	Control and Removal of Radioactive Contamination in Laboratories	Dec. 1951	.15
49	Recommendations for Waste Disposal of Phosphorus-32 and Iodine-131 for Medical Users	Nov. 1951	.15
51	Radiological Monitoring Methods and Instruments	Apr. 1952	.20
52	Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water	Mar. 1953	.25
53	Recommendations for the Disposal of Carbon-14 Wastes	Oct. 1953	.15
54	Protection against Radiations from Radium, Cobalt-60, and Cesium-137	Sept. 1954	.25
55	Protection against Betatron-Synchrotron Radiations up to 100 Million Electron Volts	Feb. 1954	.25
56	Safe Handling of Cadavers Containing Radioactive Isotopes	Oct. 1953	.15
58	Radioactive-Waste Disposal in the Ocean	Aug. 1954	.20
59	Permissible Dose from External Sources of Ionizing Radiation	Sept. 1954	.30
60	X-ray Protection	Dec. 1955	.20
61	Regulation of Radiation Exposure by Legislative Means	Dec. 1955	.25
63	Protection against Neutron Radiation up to 30 Million Electron Volts	Nov. 1957	.40

For related handbooks, see also:

NBS Handbook No.	Title	Date Issued	Price
57	Photographic Dosimetry of X- and Gamma Rays	Aug. 1954	$.15
62	Report of the International Commission on Radiological Units and Measurements (ICRU)	Apr. 1957	.40
64	Design of Free-Air Ionization Chambers	Dec. 1957	.20

recommendations, prepared as National Bureau of Standards Handbooks, are published and sold by the Superintendent of Documents, Government Printing Office, Washington, D. C. The list of the Committee's recommendations given above indicates the wide range of interests covered by its 18 working groups.

This introduction of the NCRP is primarily for the benefit of those who are not familiar with the current work of the group. However, a great number of its current activities are of immediate interest to those concerned with radiation protection and measurements as evidenced by the number of requests for information on forthcoming recommendations, and an effort will therefore be made to describe these activities in more detail than has been available in the past.*

NOTES

Addenda to H 59 and H 60 embodying the revised† (1957) maximum permissible dose levels have been issued and are available on request from the National Bureau of Standards, Publications Section, Washington, D. C. An addendum to H 54 is in the process of publication.

Revisions of Handbooks 42, 52, 54, 56, 60, and 61 are in preparation; the revision of H 56 (H 65) is expected to become available within the next few months. Announcements will be made as these and new publications are completed.

* This is the first of a continuing series of announcements of the work and programs of the NCRP that will appear in this JOURNAL.

† Maximum Permissible Radiation Exposures to Man. A Preliminary Statement of the NCRP. NBS Tech. News Bull. 1957, *41*, No. 2, 17; Taylor, L. S. The philosophy underlying radiation protection. Editorial. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1957, *77*, 910–919.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Shoreham Hotel, Washington, D. C., Sept. 30–Oct. 3, 1958.

American Radium Society
Secretary, Dr. Robert L. Brown, P.O. Box 459, Emory University, Ga. Annual Meeting: The Homestead, Hot Springs, Va., April 6–8, 1959.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Palmer House, Chicago, Ill., Nov. 16–21, 1958.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: To be announced.

Section on Radiology, American Medical Association
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: June 8–12, 1959, Atlantic City, N. J.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn. Fall 1958 Meeting and Examinations Dec. 8–12, inclusive, Washington, D. C. Final date for filing applications was July 1, 1958.

Ninth International Congress of Radiology
Secretary-General, Dr. Hans v. Braunbehrens. Request for information should be addressed to Dr. Viktor Loeck, Kongressbüro, Forsthausstrasse 76, Frankfurt am Main, Germany. Meets in Munich, Germany, July 23 to July 30, 1959.

Sixth Inter-American Congress of Radiology
Secretary for North America, Dr. Jorge de la Flor, Hospital Arzobispo Loayza, Lima, Perú. Meets in Lima, Perú, Nov. 2–8, 1958.

Alabama Radiological Society
Secretary Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

American Nuclear Society
Executive-Secretary, Dr. W. W. Grigorieff, P. O. Box 963, Oak Ridge, Tenn. Annual meeting: To be announced.

Arizona Radiological Society
Secretary, Dr. R. Lee Foster, 1313 N. Second St., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. E. A Mendelsohn, Holt-Krock Clinic, Fort Smith, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary, Dr. Paul Riemenschneider, Medical College, State University of New York, Syracuse, N. Y.

Atlanta Radiological Society
Secretary, Dr. J. L. Clements, Jr., 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

Blockly Radiological Society
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St., Philadelphia, Pa.

Brooklyn Radiological Society
Secretary, Dr. Solomon Schwartz, 555 Prospect Place, Brooklyn, N. Y. Meets first Thursday of each month October through May.

Buffalo Radiological Society
Secretary, Dr. Charles Bernstein, 685 Delaware Ave., Buffalo 9, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. Arthur R. Cohen, 41 S. Grant Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary, Dr. Arthur S. J. Petersen, 11406 Parnell Ave., Chicago 28, Ill. Meets second Thursday of each month, October to April inclusive at the Sheraton Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary, Dr. Frederick A. Rose, 2065 Adelbert Rd., Cleveland 6, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

Colorado Radiological Society
Secretary, Dr. Lorenz R. Wurtzebach, 601 E. Nineteenth Ave., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. Paul J. Kingston, 114 Woodland St., Hartford, Conn. Meets first Friday in February and April.

Dallas-Fort Worth Radiological Club
Secretary, Dr. A. H. Keene, 3707 Gaston Ave., Dallas, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Joseph O. Reed, Jr., Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Eastern Radiological Society
Secretary, Dr. John D. Osmond, Jr., Euclid-Glenville Hospital, Cleveland 19, Ohio.

Florida Radiological Society
Secretary, Dr. C. Robert DeArmas 135 Broadway, Daytona Beach, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. B. H. Malone, 1406 Reynolds St., Brunswick, Ga. Meets Spring and Fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. George P. Daurelle, Jackson Memorial Hospital, Miami 36, Fla. Meets monthly third Wednesday at 8 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Radiological Society
Secretary, Dr. Charles J. Cherre, 462 N. Taylor, St. Louis 8, Mo.

Houston Radiological Society
Secretary, Dr. Edward B. Singleton, 6621 Fannin St., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Idaho State Radiological Society
Secretary, Dr. Charles R. McWilliams, Magic Valley Memorial Hospital, Twin Falls, Idaho. Meets annually at same time as Idaho State Medical Association.

Illinois Radiological Society
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. C. A. Stayton, Jr., 313 Hume-Mansur Bldg., Indianapolis 4, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. James R. Stark, 3244 E. Douglas, Wichita, Kansas. Meets in spring with State Medical Society, and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Robert H. Akers, 1405 West Broadway, Louisville 3, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. C. P. Naidorf, 411 Parkside Ave. Brooklyn 26, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Putnam C. Kennedy, 540 N. Central Ave., Glendale 3, Calif. Meets second Wednesday of month in September, November, March, April and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Francis J. O'Connor, Augusta General Hospital, Augusta, Maine. Meets in June, September, December and March.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. James L. Booth, 899 Madison Ave., Memphis 3, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. S. F. Johnson, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. R. W. Byrne, 3321 N. Maryland Ave., Milwaukee 11, Wis. Meets monthly on fourth Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. O. J. Baggenstoss, 1953 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

Mississippi Radiological Society
Secretary, Dr. Robert P. Henderson, 316 Medical Arts Bldg., Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. Arthur T. Austin, 104 The Doctors Bldg., Billings, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Jerome Zwanger, 126 Hicksville Rd., Massapequa, L. I., N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. J. F. Kelly, Jr., 816 Medical Arts Bldg., Omaha, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. John E. Gary, 1180 Beacon St., Brookline 46, Mass. Meets third Friday of each month, October through May, at Hotel Commander, Cambridge, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Harold G. Jacobson, 210th St. and Bainbridge Ave., New York 67, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. William H. Sprunt, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. Marianne Wallis, St. Joseph's Hospital, Minot, N. D. Meetings by announcement.

North Florida Radiological Society
Secretary, Dr. Marvin Harlan Johnston, Five Points Medical Center, Jacksonville 4, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Irving Van Woert, Jr., Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. H. B. Stewart, 2920 Capital Ave., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. Francis C. Curtzwiler, 421 Michigan St., Toledo 2, Ohio. Annual meeting: To be announced.

Oklahoma State Radiological Society
Secretary, Dr. Edmond H. Kalmon, Jr., 300 Northwest 12th Street, Oklahoma City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. C. V. Allen, Veterans Hospital, Portland, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. Robert W. Hanf, 900 S. Auburn, Kennewick, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Walter P. Bitner, 234 State St., Harrisburg, Pa. Annual meeting: Harrisburg, Pa., April 24–25, 1959.

Philadelphia Roentgen Ray Society
Secretary, Dr. Roderick L. Tondreau, 3400 Spruce St., Philadelphia 4, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Edward M. Schultz, 3401 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Hotel Roosevelt.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Section, Southern Medical Association
Secretary, Dr. Ted F. Leigh, Emory University, Georgia. Annual meeting: New Orleans, La., Nov. 3–6, 1958.

Radiological Society of Greater Cincinnati
Secretary, Dr. Warner Peck, Carew Tower St., Cincinnati 2, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Jun-chu'an Wang, The Queen's Hospital

Honolulu 9, Hawaii. Meets third Monday of each month alternately at 4:30 P.M. and 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. E. H. Stratemeier, Jr., 1010 Rialto Building, Kansas City 6, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Andrew P. Dedick, Jr., 67 E. Front St., Red Bank, N. J. Meets at Atlantic City at time of State Medical Society and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr. Mario C. Gian, 610 Niagara St., Buffalo 1, N. Y. Annual meeting: To be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Harold P. Tompkins, 658 S. Westlake, Los Angeles 57, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. John W. Colgan, 277 Alexander St., Rochester 18, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Shirley Savoy Hotel, Denver, Colo., Aug. 14–16, 1958.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. Irma Smith, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at Grison's Steak House.

Section on Radiology, California Medical Association
Secretary, Dr. Nathan M. Spishakoff, 405 N. Bedford Dr., Beverly Hills, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Ralph J. Littwin, Bristol Hospital, Bristol, Conn. Meetings are held bi-monthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. Charles E. Bickham, Jr., 1835 Eye St., N.W., Washington 6, D. C. Meets Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. Annual meeting: To be announced.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. Wayne Reeser, 804 Burroughs St., Conway, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Gordon Black, Suite 2-A, El Paso Medical Center, 1501 Arizona, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. James J. Range, P.O. Box 324, Johnson City, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. J. E. Miller, 3500 Gaston Ave., Dallas 10, Texas. Next meeting January 30 and 31, 1959, Statler-Hilton Hotel, Dallas, Texas.

Tri-State Radiological Society
Secretary, Dr. Robert E. Beck, 600 Mary St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May, Service Memorial Institute.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Frank A. Kearney, II, 110 S. Curry St., Phoebus, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Wayne A. Chesledon, 306 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

West Virginia Radiological Society
Secretary, Dr. W. Paul Elkin, 515 Medical Arts Bldg., Charleston, W. Va. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Arnold Myron Wald, 406 Boston Post Road, Port Chester, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Farrell F. Golden, 5221 Tonyawatha Trail, Madison 4, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-Ray Study Club of San Francisco
Secretary, Dr. John H. Heald, 450 Sutter St., San Francisco 8, Calif. Meets monthly, third Thursday at 7:30 P.M., Children's Hospital, September through June.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá
Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá, *Secretary-General*, Dr. Roberto Calderón, Calle Central Deste No. 218, Managua, Nicaragua. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. Guillermo Santín. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 86, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Empire

Association of Radiologists of the Province of Quebec
Secretary, Dr. Isadore Sedlezky, 3755 Cote St. Catherine Rd., Montreal, Que. Meets four times a year.

British Institute of Radiology Incorporated with the Röntgen Society
Honorary Secretary, Dr. John Blewett, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Guillaume Gill, *Associate Honorary Secretary*, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 25, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Louis Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. Arthur B. Sullivan, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

O Colégio Brasileiro de Radiologia
Secretary-General, Dr. Walter Bomfins Pontes. Seat: Rua Major Quedinho, 99-2°. Andar, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Juan L. Ambrogna, Montevideo 745, Buenos Aires. Meetings are held monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiologia
Secretary, Dr. Armando Doberti, Merced 565, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejia Dzg., Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuadoriana de Radiologia y Fisioterapia
Secretary, Dr. Enrique Ortega Guzmán, Casilla 4719, Guayaquil, Ecuador.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos G., Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Roberto François, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Sebastian Nuñez Mier y Téran, Apartado No. 9362, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Austrian Roentgen Society
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Électroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France. National Meeting Oct. 15–17, 1958 in Paris.

Ceskoslovensk společnost pro Röntgenologii a Radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut, Lagenbeckstr. 1, Mainz, Germany. Meets beginning of Oct., 1958 in Bremen. President of Congress, Professor Dr. du Mesnil de Rochemont, Universitäts—Strahlenklinik, Marburg, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiologia y Electrologia
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgengesellschaft (Société Suisse de Radiologie)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The next list of Meetings of Radiological Societies will be published in the September issue of the Journal.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Tumors of the Soft Somatic Tissues. By George T. Pack, M.D., LL.D., F.A.C.S., Attending Surgeon, Memorial Center for Cancer and Allied Diseases; Associate Professor of Clinical Surgery, Cornell University Medical College; Surgeon, Pack Medical Group, New York; and Irving M. Ariel, M.D., F.A.C.S., Associate Clinical Professor of Surgery and Associate Attending Surgeon, New York Medical College, Flower and Fifth Avenue Hospitals; Surgeon, Pack Medical Group, New York. Cloth. Price, $30.00. Pp. 820, with 652 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1958.

This excellent volume, the first encompassing text to deal with the organs of locomotion and support, fulfills a long-standing need in the library of the oncologist. Perhaps the most valuable aspect of this book in addition to its completeness is its recording of the vast personal experiences of the authors and their collaborators. Certain sections have ample bibliographies, but for the most part a minimal review of the literature complements the evaluations of their own experience.

There is an excellent introductory section on the nosologic aspects of tumors of the soft somatic tissues. Lesions originating from parenchymal and skeletal structures are excluded. Diseases of the lymphoid system, traditionally treated as a separate group, are also excluded as are diseases arising in the bone marrow and from the skin. Due to the scope of dealing with melanomas, this problem will form the basis for a subsequent volume. Neoplasms arising from the peripheral nerves are considered because of their similarity to soft tissue tumors. Section I of the book, in addition to nosology, includes discussions of the incidence, hereditary and congenital factors, etiologic relationships of trauma, incidence of malignant change in benign lesions, and the proclivity of local lesions for regional lymph node spread. Insofar as trauma is concerned, irradiation is by far the most important injurious agent. External physical effects and inherent tissue predisposition to the effects of trauma are infrequently observed as etiologic factors. Benign lesions become malignant infrequently, but the incidence of metastases to the lymph nodes is greater than the traditional concept of sarcomatous spread being principally hematogenous as opposed to carcinomatous spread being predominantly lymphogenous. Any sarcoma must be suspected of being capable of metastasizing to regional lymph nodes in planning a course of treatment.

Section II deals with the general principles of treatment, assessing the factors of age, the general condition of the patient and the philosophy bearing upon the treatment of localized distant metastases from sarcomata. A timely chapter on the technique of various biopsy procedures is included. The indications and technique of local excision, excision and dissection, and amputations of varying extents are discussed. Hip joint disarticulation, sacroiliac disarticulation and interscapulothoracic amputation are each separately presented due to infrequency of application and complexity. The last portion of this section on general treatment policies deals with radiation therapy in the light of the radiosensitivity or radiocurability of various lesions. The numerous techniques employed in delivering radiation therapy are then described. Of particular interest is the conversion of certain inoperable conditions to an operable status. In general, it is stated that radiation therapy is of principal use in this group of diseases as an adjunct to surgery but should a contraindication to surgical treatment exist, an attempt to cure should be made utilizing irradiation techniques.

Section III is divided into chapters dealing with tumors of fibrous tissue, primitive mesenchyme, osteoblasts and chondroblasts, adipose tissue, blood and lymph vessels, synovial tissues, smooth muscle, striated muscle and peripheral nerve structures. In the first group on fibrous tissue tumors, the concept of a desmoplastic diathesis as a general connective tissue response on the part of the host to a malignancy and the possible relationship of some entities in this category to the collagen diseases is of particular interest. Lesions deriving from the primitive mesenchyme are divided into

mesenchymomas and myomas. The osteoblastic and chrondroblastic lesions are discussed only as they occur in the soft tissues. The chapter dealing with blood and lymph vessel lesions presents an excellent classification and is illustrated with outstanding case material. No less than 28 synonyms are listed for Kaposi's hemorrhagic sarcoma.

Section IV describes the soft somatic tissue tumors in infancy and childhood and corrects certain misconceptions as to their curability, incidence, growth characteristics and clinical stage at the time of diagnosis. A more optimistic view is taken toward this group of tumors than is usually presented.

Section V considers the therapeutic management of certain neoplasms on a regional basis, those arising in the head and neck, abdominal wall, retroperitoneal space and the buttocks. Those involving the abdominal wall are particularly of interest in view of their wide spread lymphatic ramifications. The retroperitoneal space is all too often an area of confusion, but the authors have carefully classified the various lesions arising here and emphasize the great variety of tumors which can occur in this location. Sarcoma of the buttocks is separately considered due to regional characteristics.

Section VI presents graphically the end results of the case material forming the basis of this text. An evaluation is made of various factors that influence prognosis and the lesions are generally classified into determinate and indeterminate groups. The survival rates of certain more commonly encountered lesions are also given graphically. An over-all five year survival of 39 per cent is reported by the authors, a figure which is accomplished no doubt by painstaking attention to the manifold problems involved in managing these lesions.

This book will become an outstanding reference work for this group of diseases. The authors have done an excellent service to all of the disciplines in medicine charged with the diagnosis and treatment of these difficult lesions. It should become an integral part of the bookshelf of the surgeon and the radiation therapist.

K. L. Krabbenhoft, M.D.

La Broncografia. By L. di Guglielmo, Istituto di Radiologia dell'Università di Pavia; L. Pigorini, Istituto "C. Forlanini" Clinica Tisiologica dell'Università di Roma; and G. A. Citroni, Clinica Otorinolaringoiatrica dell'Università di Pavia. Cloth. Pp. 674, with 621 illustrations. Il Pensiero Scientifico Editore, Via Brenta 13, Rome, Italy, 1957.

This imposing volume is the result of the collaboration of two radiologists and one otolaryngologist who have performed 4,000 bronchographies with a matchless technique. In the opinion of the reviewer, this book is one of the most complete ever published on a subject of timely interest to all radiologists. The descriptive text is clear and all illustrations are superbly reproduced in large size.

The book is divided into three parts. The first part includes ten chapters devoted to the technique of bronchography and to the anatomy and physiology of the bronchial tree. The second part comprises seven chapters in which the roentgenographic signs of the normal and abnormal bronchi are thoroughly discussed. The third part contains 27 chapters dealing with various pathologic conditions and is illustrated with roentgenograms as well as with excellent color reproductions of the bronchoscopic findings. In these last 27 chapters the authors discuss malformations of the bronchial tree, inflammatory diseases, pneumonitis, bronchiectasis, lung abscess, tuberculosis, syphilis, mycoses, primary and secondary malignancies, bronchial adenoma, asthma, emphysema, echinococcus cyst, pneumoconioses, foreign bodies and traumatic lesions. The bronchial displacements following pneumothorax, the pleural effusions, diseases of the mediastinum, lesions of the thoracic wall, surgical procedures and irradiation techniques are also discussed and illustrated.

A book as complete and as well organized as this one should be read in the full text. It is to be hoped that the authors will find it possible to publish an English translation of their work to enable English and American workers to benefit from their vast experience with bronchography.

Cesare Gianturco, M.D.

The Lung as a Mirror of Systemic Disease. By Eli H. Rubin, M.D., Professor of Clinical Medicine, Albert Einstein College of Medicine, Yeshiva University; Director of Pulmonary Diseases, Bronx Municipal Hospital Center; Attending Physician, Division of Pulmonary Diseases, Montefiore Hospital; and Consulting Physician in Pulmonary Diseases, Lebanon and Morrisania City

Hospitals, New York, New York. Cloth. Price, $12.50. Pp. 288, with 92 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

This monograph, written by an internist who has made a study of pulmonary lesions, is devoted to the description of a number of diseases which are systemic in character and which produce either nonspecific or more or less characteristic roentgen changes. Diseases which are strictly pulmonary in their primary importance are not included; and the discussion emphasizes some of the less commonly thought of conditions in which pulmonary changes are seen.

The material is well organized into chapters, dealing with diseases of metabolism, diseases of the blood, allergic diseases, diseases of the skin and mucous membranes, and cardiovascular diseases. There are additional important chapters on mass chest surveys, diagnosis of diseases of the chest, either primary or as part of systemic disease, and a discussion of the technique and pitfalls of roentgen examination of the chest.

The presentation is from the point of view of the internist, and each disease is considered from its historic background, the clinical features, which include differential diagnosis, laboratory aids in diagnosis, pathology, which includes pathologic physiology, and roentgenology. There are also a few comments on treatment. The author has avoided a condensed style, and the discussions are all punctuated by numerous examples, many obtained from case reports found in the literature. There is a number of more formal case reports supplied by the author. The work is very well documented throughout, and there is an excellent bibliography at the end of each chapter.

Since this is primarily for the nonradiologist, the chapters on techniques and pitfalls of roentgen examination of the chest are very important. The author stresses some of the limitations inherent in roentgen study which are always present in the mind of the radiologist, and which the referring physician is prone to forget. The nonspecific character of roentgen changes is emphasized, and it is pointed out that clinical and laboratory information, particularly the patient's history, are used by the experienced radiologist, and are of utmost importance in evaluating the roentgen findings to the best advantage of the patient. At the same time the author makes it clear that study of the patient cannot be divided into clinical, roentgenologic or laboratory divisions, but that each bit of information modifies and elucidates the rest, and all are not separate parts but a whole.

This volume is not an attempt to teach radiology to physicians, but it is an attempt to show the value of chest roentgenography in some cases where this might be overlooked. For this reason it should be read by the radiologist as part of his background. It holds very important information for the general practitioner and the internist. There is also a chapter covering abdominal diseases which would be of interest to the surgeon.

One minor fault which some radiologists might find is the tendency to describe the roentgen findings by characterizing the type of shadow rather than naming the pathologic process: thus fibrosis or lobular atelectasis becomes linear density or interstitial striation; emphysema, either bullous or diffuse, becomes hyperillumination. The author has not attempted an exhaustive treatise, but the fine arrangement and the good index make the material easy to find, and the excellent bibliography makes available more than enough material to satisfy anyone whose questions are not completely answered.

E. F. Lang, M.D.

Bronchopulmonary Diseases; Basic Aspects, Diagnosis and Treatment. Edited by Emil A. Naclerio, M.D., Chief of the Thoracic Surgical Services, Harlem and Columbus Hospitals, New York. Cloth. Price, $24.00. Pp. 956, with 719 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1957.

This text of almost 1,000 pages is actually a publication in book form of a series of 142 papers by men who have particular interest and knowledge of the subjects on which they write. These men of many specialties, bring together material that otherwise would be divided among many specialty journals in publication.

Excellent editing and selection has resulted in an exceptionally complete and logical coverage of a very broad subject. The degree to which the fundamentals of embryology, anatomy, physiology and development are covered is unusual in a text which also covers the diagnosis and treatment of disease. Correlation of the clinical aspects of disease with basic fea-

tures points up the importance of such information. The methods of diagnosis are discussed in a series of papers, including not only the older and widely employed tests and examinations, but also the use of physiologic tests, such as lung volume measurements and ventilation movements, the uses of the various types of endoscopic examinations, and methods of biopsy. The indications for, reliability of, and hazards associated with, the various procedures are well presented.

In general throughout the text, terminology is carefully and specifically defined.

The sections dealing with roentgen procedures constitute approximately 75 pages, but diagnosis and differential diagnosis by roentgen examinations are included in the majority of the other papers. Thus, from the scope of the roentgenologic coverage alone, this book is of interest and value to the roentgenologist, but more importantly, the correlation with the current concepts of physiology, normal and pathologic, and management of disease, is excellent.

Extremely practical are the discussions of lung disease secondary to heart disease, and vice versa, and the differential diagnosis between heart and lung insufficiency.

The relationship of nose and throat to pulmonary disease is the subject of one section.

The diseases of the lungs are presented separately, with coverage of diagnosis, pathology, and specific methods of treatment, and in addition, a separate 50 page section deals with fundamental considerations in management. This discusses respiratory function, inhalational therapy, corticotrophin and corticosteroids, and anesthesia in the patients with altered pulmonary function and tracheotomy. The selection and management of patients for air travel is also given consideration.

Surgery and anesthesia in bronchopulmonary disease are discussed in considerable detail. The sections included with treatment of tuberculosis are particularly specific.

Although developmental abnormalities and tension problems in infants and children are well covered, and in tuberculosis the treatment of this disease in childhood is a separate topic, the common inflammatory and infectious diseases of children are not specifically considered, nor are the chemical pneumonias, such as those associated with ingestion of hydrocarbon derivatives.

Management of malignant disease of the lung by chemotherapy, radioactive isotopes, radiation therapy, and supportive medical treatment is described.

It is impossible in review of such a book to summarize the content or even adequately appraise it with any degree of completeness. The wide scope makes even a sketchy comment on the many excellent papers impractical. As would be expected with this type of text, there is a little overlap in some of the papers, but judicious selection and editing has kept this to a minimum, and has resulted in a remarkably complete and coherent coverage. The index is very good, permitting its use for reference, but this is a very readable book and initial reading in continuity will insure optimum value for reference purposes. There are few branches of medicine in which this book would not be a definite asset to the library.

Lois Cowan Collins, M.D.

BOOKS RECEIVED

Our Nuclear Adventure; Its Possibilities and Perils. By D. G. Arnott. Cloth. Price, $6.00. Pp. 170, with 17 illustrations. Philosophical Library, Inc., 15 East 40th Street, New York 16, New York, 1958.

The Treatment of Fractures. Volume III. By Lorenz Böhler, M.D., Director of Accident Hospital, Vienna; Professor of Accident Surgery, University of Vienna. Translated from the Thirteenth German Edition by Alfred Wallner, M.D., Diplomate, American Board of Surgery, and Otto Russe, M.D., Director of the Accident Hospital, Vienna. Cloth. Price, $21.00. Pp. 2,307, with 1,699 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1958.

The Atomic Age and Our Biological Future. By H. V. Brønsted; translated by E. M. Huggard. Cloth. Price. $4.75. Pp. 80. Philosophical Library, Inc., 15 East 40th Street, New York 16, New York, 1957.

The Medical Management of Cancer. By Henry D. Diamond, M.D., F.A.C.P., Associate Attending Physician, The Medical Neoplasia Service of the Department of Medicine, The Memorial Center for Cancer and Allied Diseases; Associate, and Section Head of the Lymphoma Section of the Division of Clinical Chemotherapy, The Sloan-Kettering Institute for Cancer Research; Assistant Professor of Clinical Medicine, Cornell University Medical College, New York, N. Y. Cloth. Price, $6.75. Pp. 179, with 40 illustrations and 23 tables. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1958.

Bone and Radiostrontium. By Arne Engström, Rolf Björnerstedt, Carl-Johan Clemedson, and

Arne Nelson. Cloth. Price, $8.75. Pp. 139, with numerous illustrations. John Wiley & Sons, Inc., 440 Fourth Avenue, New York 16, New York; Almqvist & Wiksell, Stockholm, Sweden, 1958.

THERE IS AN ANSWER TO CANCER. By Leonard B. Goldman, M.D. Cloth. Price, $3.50. Pp. 178. Harper & Brothers, 49 E. 33rd Street, New York 16, New York, 1958.

ATOMIC ENERGY IN MEDICINE. By K. E. Halnan. Cloth. Price, $6.00. Pp. 157, with 31 illustrations and 11 tables. Philosophical Library, Inc., 15 East 40th Street, New York 16, New York, 1957.

DYNAMIC ANATOMY AND PHYSIOLOGY. By L. L. Langley, Ph.D., LL.B., Associate Professor of Physiology, University of Alabama Medical Center; E. Cheraskin, M.D., D.M.D., Professor and Chairman of the Department of Oral Medicine, University of Alabama Medical Center; and Ruth Sleeper, R.N., Director of the School of Nursing, Massachusetts General Hospital. Cloth. Price, $6.00. Pp. 719, with 313 illustrations. McGraw-Hill Book Company, Inc., 330 West 42nd Street, New York 36, New York, 1958.

ÉTUDE RADIOLOGIQUE DE LA CIRCULATION VEINEUSE DU MEMBRE SUPÉRIEUR (SUJET NORMAL). By Ercole Lavizzari and Valerio Ottolini. Paper. Price, 2,400 fr. Pp. 122, with 38 illustrations. Masson et Cie, Éditeurs, 120 boulevard Saint-Germain, Paris 6e, France, 1958.

CLINICAL ENZYMOLOGY. Edited by Gustav J. Martin, Sc.D., Research Director, The National Drug Company, Philadelphia. Cloth. Price, $6.00. Pp. 241, with figures and tables. Little, Brown & Company, 34 Beacon Street, Boston 6, Massachusetts, 1958.

WESEN UND BEDEUTUNG DER ENCHONDRALEN DYSOSTOSEN. By Priv.-Doz. Dr. H. Mau, Heidelberg. Paper. Price, DM 27.—. Pp. 219, with 83 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

LOCAL ANAPHYLACTIC CHANGES IN RABBIT EARS; WITH SPECIAL REFERENCE TO ANAPHYLACTIC HYPERKERATOSIS AND PAPILLOMATOSIS AND A SURVEY OF THE MECHANISM. Acta dermato-venereologica, Volume 36, Supplement 36. By O. Reuterwall. Paper. Acta dermato-venereologica, Karolinska Sjukhus, Stockholm 60, Sweden, 1956.

ANNUAL REVIEW OF MEDICINE. VOLUME 9. 1958. David A. Rytand, Editor. Cloth. Price, $7.00. Pp. 530, with illustrations and tables. Annual Reviews, Inc., Stanford, California, 1958.

MEDIZINISCHE RÖNTGENTECHNIK. II. PHYSIKALISCH-TECHNISCHER TEIL. Edited by Prof. Dr. Herbert Schoen, Karlsruhe. Second edition. Cloth. Price, DM 48.—. Pp. 463, with 281 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

RADIOTERAPIA MODERNA. By Prof. Félix Wachsmann, Prof. Gunther Barth, and Dr. Sabino Di Rienzo. Cloth. Pp. 313, with 129 illustrations. Editorial Assandri, Dean Funnes 61, Córdoba, Republica Argentina, 1957.

TREATMENT OF MALIGNANT BLOOD DISEASES BY RADIOACTIVE PHOSPHORUS; PART I. CLINICAL ASPECTS. Acta radiologica, Supplement 150. By Ingmar Bergström and Erik Lindgren. Paper. Price, Sw. Kr. 25:-. Pp. 107, with 23 illustrations and 10 tables. Acta radiologica, Stockholm 2, Sweden, 1957.

TREATMENT OF MALIGNANT BLOOD DISEASES BY RADIOACTIVE PHOSPHORUS; PART II. HEMATOLOGICAL ASPECTS. Acta radiologica, Supplement 150. By Ingmar Bergström. Paper. Price, Sw. Kr. 25:-. Pp. 98, with 69 illustrations. Acta radiologica, Stockholm 2, Sweden, 1957.

RADIOLOGIC EXAMINATION OF THE BRAIN AND SPINAL CORD. Acta radiologica, Supplement 151. By Erik Lindgren. Paper. Price, Sw. Kr. 35:-. Pp. 147, with 178 illustrations. Acta radiologica, Stockholm 2, Sweden, 1957.

PRIMARY TUMORS OF THE PELVIC BONES; A ROENTGEN DIAGNOSTIC STUDY OF EIGHTY-THREE CASES. Acta radiologica, Supplement 152. By Karl Gustav Helander and Åke Lindbom. Paper. Price, Sw. Kr. 25:-. Pp. 62, with 48 illustrations. Acta radiologica, Stockholm 2, Sweden, 1957.

ÜBER DAS VORKOMMEN VON HIATUSHERNIEN UND KARDIAINSUFFIZIENZ IN VERBINDUNG MIT PLEURASCHWARTE. Acta radiologica, Supplement 153. By Otso Kuosmanen. Paper. Price, Sw., Kr. 20:-. Pp. 76, with 19 illustrations. Acta radiologica, Stockholm 2, Sweden, 1957.

THE HISTOLOGICAL DISTRIBUTION OF PROTEINASE AND PEPTIDASE ACTIVITY IN SOLID TUMOR TRANSPLANTS. Acta radiologica, Supplement 154. By B. Sylvén and H. Malmgren. Paper. Price, Sw. Kr. 30:-. Pp. 124, with 19 illustrations and 7 tables. Acta radiologica, Stockholm 2, Sweden, 1957.

ANGIONEPHROGRAPHY AND SUPRARENAL ANGIOGRAPHY. Acta radiologica, Supplement 155. By Gunnar Edsman. Paper. Price, Sw. Kr. 25:-. Pp. 141, with 67 illustrations and 18 tables. Acta radiologica, Stockholm 2, Sweden, 1957.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Schmidt, H. Röntgenologische Darstellung eines Processus retromastoideus. (Roentgenologic demonstration of a retromastoid process.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Jan., 1958, *88*, 67–69. (From: Medizinisches Strahleninstitut der Universität, Tübingen, Germany.)

The retromastoid process arises from the junction between the superior and inferior limb of the nuchal crest behind the mastoid process and at the insertion of the superior oblique occipital muscle. It forms a blunt, sometimes rounded process 0.5 by 2 cm. in size and can be uni- or bilateral. Some investigators regard this formation as a residuum of an occipital vertebra.

The retromastoid process is noted more frequently in the skulls of Polynesians. In the presented case, a white woman, it was found accidentally in anteroposterior projection of the cervical spine following an injury. The laminagraphic studies did not show any indication for existence of an occipital vertebra and, therefore, the author is of the opinion that his case represents a true retromastoid process of the right side. Five illustrations are included.—*Nathan Kriss, M.D.*

Jeschek, J., and Muntean, E. Die Schichtuntersuchung des fenestrierten Labyrinthes. (Laminagraphy of the fenestrated labyrinth.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1957, *87*, 512–517. (Address: E. Muntean, Bischofplatz 1, Graz, Austria.)

Laminagraphy of the labyrinth after fenestration operation is a very valuable method to rule out secondary closure of the window and is used to determine the indications for reoperation.

Technique: The patient's head is inclined 45° toward the affected side (Stenver) and the first layer is placed at the posterior attachment of the auricle. Usually no more than 2–3 exposures are necessary (Muntean's technique).

Five cases are discussed and illustrated.—*Frederick M. Reis, M.D.*

Wood, Ernest H., and Farmer, Thomas W. Cerebral infarction simulating brain tumor. *Radiology*, Nov., 1957, *69*, 693–698. (Address: E. H. Wood, School of Medicine, University of North Carolina, Chapel Hill, N. C.)

The deprivation of blood to the brain, or cerebral infarction, usually results in atrophy of the affected tissue and a displacement of intracranial contents toward the affected side. However, during the early stages of an infarction, edema may ensue and the affected portion of the brain may expand and produce clinical and roentgenologic signs simulating brain tumor. Thus an early infarction should be considered when confronted with these signs. The edema of an infarcted hemisphere with contralateral shift of intracranial contents may be manifested roentgenologically by: (a) shift of the pineal gland; (b) dislocation of the lateral and third ventricles on pneumography; (c) displacement of the anterior cerebral artery (if filled) on angiography. As recent studies indicate that internal carotid artery thrombosis may be more common than was formerly appreciated, carotid angiography is indicated in patients where the differential diagnosis between cerebral tumor and cerebral vascular occlusion cannot be determined by other means.

The authors report 4 cases which presented clinical and roentgenologic evidence of brain expansion as a result of arterial thrombosis.—*John Alexander, M.D.*

Paglicci, A., and Marzano, E. Metastasi cerebrali da tumore pulmonare primitivo. (Cerebral metastases from primary cancer of the lung.) *Radiol. med.*, March, 1958, *44*, 277–283. (From: Istituto di Radiologia Medica dell'Università di Roma, Rome, Italy.)

This is a report on 10 cases of metastases to the brain from a series of 152 patients with primary carcinoma of the lung, observed during the years 1947–1957, in the Department of Radiology of the Medical School of the University of Rome.

The clinical, roentgenologic and pathologic findings are reviewed.—*A. F. Govoni, M.D.*

Neck and Chest

Rosenthal, Ira M. The pneumonias of childhood. *G.P.*, March, 1958, *17*, 77–87. (From: Department of Pediatrics, University of Illinois College of Medicine, Chicago, Ill.)

In this excellent general survey, the author stresses that the pneumonias of childhood are a heterogenous group and generally due to one of many organisms, although they may be noninfectious. The characteristic symptoms of pneumonia as seen in adults are also found in children which include fever, cough, chest pain, anorexia, malaise and rapid respiration. However, productive cough is seldom seen in younger children and the respiratory rate tends to show a greater increase over normal than in adults. Also, intercostal and subcostal retractions and flaring of the alae nasae are more frequent in young children than in adults. In addition, gastrointestinal symptoms are commonly noted in infants and young children at the onset of the disease. The physical signs in children tend to be less definite than the physical signs in adults and the physician must rely more on the general appearance of the patient. In the

pathologic classification, pneumonias of children may be divided into alveolar and interstitial pneumonias. In young children under the age of two, alveolar pneumonias tend to be lobular in distribution, thus rarely giving massive consolidation. In the interstitial pneumonias, inflammation of the bronchial and bronchiolar walls results in obstructive atelectasis, emphysema or both. These tissue changes give the usual mottled appearance of lung fields on the roentgenogram. Classified according to etiology, most of the pneumonias of childhood are infectious in origin. A miscellaneous group including aspiration of chemical agents such as kerosene or zinc stearate, rheumatic pneumonias, plasma cell pneumonia and eosinophilic pneumonia constitutes a small segment of childhood pneumonias.

The author reviews in serial fashion the bacterial pneumonias of childhood indicating the antibiotic of choice and average dosage. He indicates the possible complications in each group and the prognosis. In addition, the general therapeutic measures such as rest, light diet, maintenance of good hydration, oxygen with adequate humidification and blood transfusions are all listed as necessary adjuncts to good therapy.

In conclusion, the author stresses that although there have been remarkable advances in therapy of most bacterial pneumonias, there is no place for complacency in the management of the pneumonias of childhood. While chest roentgenograms and clinical studies are of great value, careful planning of the therapy and attention to general nonspecific measures is essential for effective management.—*T. D. Allison, M.D.*

Chiariotti, Franco. Quadri radiologici del pneumatocele e della sua evoluzione. (Radiologic aspects of pneumatocele and its development.) *Radiol. med.*, March, 1958, *44*, 209–224. (From: Reparto Radiologico dell'Ospedale di Busto Arsizio, Italy.)

The term pneumatocele adopted by the author refers to the appearance of a cyst or cysts during an acute respiratory infection. The main feature of these pneumocysts, which is very important from the differential diagnostic point of view, is their disappearance after the clearing of the pneumonic process.

The generally accepted hypothesis on the formation of these cysts is the one which admits the presence of a bronchial expiratory valve secondary to a narrowing of the bronchial lumen, resulting from an inflammatory process. Caffey calls these cysts "regional obstructive emphysema."

The diagnosis of these cysts can be made only by roentgenographic examination and their recognition is very important in order to establish the most adequate therapy.

The most common complication observed in the cases reported by the author was the rupture of the cyst, when it was situated near the surface of the lung, with subsequent appearance of a pneumothorax.

In one case only, the cyst became infected but in this case also, five months after the onset of the disease, a roentgenographic examination showed a complete "restitutio ad integrum."—*A. F. Govoni, M.D.*

Keats, Theodore E., and Koenig, George F. Magnification technic in roentgenography of the chest. *Radiology*, Nov., 1957, *69*, 745–747. (Address: T. E. Keats, University of Missouri, Columbia, Mo.)

The advent of the 0.3 mm. focal spot roentgen tube has, in recent years, popularized the magnification technique in roentgenography. In the past, this technique has been used chiefly for the magnification of skeletal parts. The authors here propose its use for the investigation of poorly defined parenchymal lesions in the chest.

The technique is described and illustrative roentgenograms are presented. Although the method may be employed successfully in adults, the authors believe that its greatest usefulness is in pediatric chest roentgenography.—*Z. Petrany, M.D.*

Fischer, Harry W., Lucido, Joseph L., and Lynxwiler, Chester P. Lobar emphysema. *J.A.M.A.*, Jan. 25, 1958, *166*, 340–345. (From: The Department of Radiology, State University of Iowa; the Department of Surgery and the Department of Pediatrics, St. Louis University School of Medicine, St. Louis, Mo.)

The authors report the case of a newborn infant with unilobar emphysema on the second day of life and describe the clinical, roentgenographic and pathologic findings and the therapy.

The symptoms consist of severe respiratory difficulty with cyanosis, unrelated to acute infection or other known event. Cough and stridor are sometimes observed but are relatively insignificant compared to the primary disturbance of respiratory exchange. The physical examination reveals labored breathing, cyanosis and signs of hyperaeration of a unilateral area or entire hemithorax. The heart is shifted away from the hyperresonant side. The poorer breath sounds are heard on the same side as the hyperresonant percussion note. Chest roentgenograms show a highly radiolucent lobe area, or entire lung field still containing pulmonary markings. Other lung tissue appears poorly aerated and individual lobes may be compressed or collapsed. The ipsilateral leaf of the diaphragm is compressed and the mediastinal contents are shifted away from the radiolucent side. The chest diaphragmatic movements are more active on the poorly aerated, less radiolucent side. Usually no

lesion is seen through the bronchoscope, but occasionally abnormal collapsibility of the bronchial walls may be noted.

The patient described presented the above symptoms and physical findings. Thoracotomy was performed on the seventh day of life revealing a hugely distended right middle lobe under tension pushing the heart and thymus gland to the left. The right upper and lower lobes were partially collapsed. Some difficulty was encountered in moving air out of the right middle lobe through the middle lobe bronchus. A right middle lobectomy was performed without difficulty. At the conclusion of the procedure, the upper and lower lobes expanded well. The postoperative course was uneventful and the remaining tissue in the right lung appeared well expanded. The pathologic examination of the right middle lobe did not demonstrate any abnormality of the bronchial wall cartilages. Cyst formation was not present and the emphysematous process involved the entire lobe.

The child was observed at the age of six months and no further respiratory difficulty had occurred. Chest roentgenogram was within normal limits.

Unilobar emphysema appears to be an uncommon disease with an apparent increase in incidence in recent years which is probably due to two factors. The first is wider use of chest roentgenography and fluoroscopy for the young infant and secondly, awareness of the possibility of such a disease results in a diagnosis being made more frequently by the radiologist, the pediatrician, or the surgeon.

Unilateral emphysema can neither be recognized nor excluded on the basis of physical examination alone. Anteroposterior and lateral chest roentgenograms are essential for the diagnosis. Certain lesions producing the same symptoms show roentgenographic findings similar to those of lobar emphysema. These comprise regional atelectasis, with contralateral compensatory emphysema; cystic lung disease, including giant solitary cyst and pneumatocele; pneumothorax; obstructive emphysema secondary to intrabronchial foreign bodies; diaphragmatic hernia; tracheoesophageal fistula; and agenesis of the lung. The presence of pulmonary markings in the radiolucent area differentiates lobar emphysema from pneumothorax, localized cystic lung disease, solitary giant cyst, and diaphragmatic hernia.

There are two paramount points in the diagnosis. Persistence of lung markings in the large radiolucent area, whether this be all or part of the unilateral lung field, is highly important. The collapsed right lower lobe will appear as a triangular density adjacent to the right lower border of the heart. The second prime diagnostic point is information obtained from bronchoscopy and the response of the patient to tracheobronchial aspiration, by which the more common atelectasis—compensatory emphysema complexes can usually be excluded.

About one-third of the reported cases of lobar emphysema have been secondary to an abnormality of bronchial cartilage. Anatomically there is an absence or malformation of one or more cartilage rings of the lobar bronchus. Although cartilage anomaly is the most commonly demonstrated cause of lobar emphysema, extrabronchial compression by an anomalous vessel or by redundant intrabronchial mucosal folds or shelves has been known to act in the same check-valve fashion to produce unilobar emphysema. Another possible etiology is that lobar emphysema may result from a mucus plug exerting a check-valve effect in the lobar bronchus. Absence or deficiency of elastic tissue in the lobe has not yet been demonstrated.

The treatment will always depend on the severity of the symptoms and the threat to life. Exploratory thoracotomy is recommended for the patient with lobar emphysema when his initial severe symptoms do not respond quickly and well to the administration of oxygen and to tracheobronchial aspiration by catheter and bronchoscope and when the roentgenologic findings remain unchanged or only minimally improved. There is evidence that some infants with lobar emphysema can be tided over their acute symptoms by administration of oxygen, humidification of inspired air, tracheobronchial aspiration and other conservative measures. These children gradually return to their more or less normal state with disappearance of the roentgenologically demonstrated emphysema over a variable period of time.

Attempts to relieve the localized emphysema by introduction of needle through the chest wall has mainly been unsuccessful and has been condemned by several authors because of the production of a tension pneumothorax.

The authors collected from the literature 36 patients who have been greatly benefited by surgery. Thirty-one have had no further respiratory difficulties and are living a normal life.—*James F. Martin, M.D.*

Harders, H., and Meier-Siem, M. Gleichzeitiges Vorkommen der sogenannten tiefen Rechtslage der Brustaorta bei eineiigen Zwillingen. (Simultaneous occurrence of the so-called deep right-sided deviation of the thoracic aorta in uni-ovular twins.) *Radiol. clin.*, July, 1957, *26*, 187–198. (From: Medizinische Klinik und Strahleninstitut des Universitätskrankenhauses, Hamburg-Eppendorf, Germany.)

Deviation of the descending aorta to the right with impingement on the esophagus is described for the first time in uni-ovular twins. The condition is due to arteriosclerotic elongation of the aorta. A congenital or inherited factor which determines the specific location of the sclerotic changes is stipulated. A congenital malformation is not responsible for this change.

History, roentgen appearance, mechanism of ori-

gin, as well as the differentiation from congenital malformations and mediastinal lesions, are discussed and the authors' own case presented.—*Frederick M. Reis, M.D.*

Lester, Richard G., Gedgaudas, Eugene, and Rigler, Leo G. Method of radiologic diagnosis of congenital heart disease in children. *J.A.M.A.*, Feb. 1, 1958, *166*, 439–443. (Address: R. G. Lester, 412 S. E. Union St., Minneapolis 14, Minn.)

The possibility of surgical correction of many congenital heart lesions has emphasized the significance of precise diagnosis. Roentgenologic evaluation of congenital heart lesions is the most important single procedure available. The purpose of this paper is to suggest a method of analysis of fluoroscopic and roentgenographic findings. The approach is based on a study of the roentgenologic manifestations of the pathologic anatomy and physiology of these lesions and is an attempt to co-ordinate the respective findings.

The usual classification of congenital heart disease into cyanotic and acyanotic types separates the patients into great physiologic groups. There are, however, several disadvantages to this classification from the clinical, and particularly from the roentgenologic, point of view. First, not all the patients with peripheral desaturation are clinically cyanotic. Second, some patients with acyanotic heart disease may be cyanotic as a result of failures of other mechanisms. Finally, cyanosis is not a roentgenologic criterion.

The authors have devised a classification that permits an objective division of congenital heart disease into 3 groups on the basis of roentgenologic criteria. They are divided into groups according to the pulmonary arterial vasculature; *i.e.* increased, normal and decreased pulmonary arteries.

All patients with *decreased* pulmonary vasculature are peripherally desaturated and are in the cyanotic group. All the patients in this group have shunts from the right side of the heart into the systemic circulation. The most common lesion is the tetralogy of Fallot. Tricuspid atresia and another considerably less common lesion, triology of Fallot, are also in this group.

Patients with *normal* pulmonary vasculature have full saturation and are in the acyanotic group. The most common lesion in this group is coarctation of the aorta. Aortic stenosis, congenital mitral stenosis, cor triatriatum, pure pulmonary stenosis, aortic ring, hereditary sclerosis, spinal ataxia (Friedreich's ataxia), progressive spastic paraplegia resulting from hereditary syphilis (Marfan's disease), subendocardial fibroelastosis, congenital ventricular aneurysm, anomalous origin of the left coronary artery, glycogenesis (von Gierke's disease), cardiac tumor, hypertensive, metabolic and rheumatic heart disease also fall within this group, although all are not congenital in origin. Isolated valvular pulmonic stenosis in the late stages, may show a relative or even absolute decrease in the size of peripheral pulmonary arterial vessels but it is important to remember that in most cases the pulmonary vasculature in this disease is normal.

Patients with *increased* pulmonary vasculature have been divided into 3 subgroups: (1) In this subgroup, by far the largest, the patients have increased pulmonary vasculature as a result of an intracardiac or extracardiac left or right shunt. Usually, there is no peripheral desaturation and no cyanosis. Interatrial septal defect, interventricular septal defect, atrioventricularis communis, patent ductus arteriosus, partial anomalous pulmonary venous return, aortic-pulmonic window, ruptured sinus of Valsalva, corrected transposition with left to right shunt, and atrial septal defect with mitral stenosis and dilatation of the pulmonary artery may be found in the patients in this subgroup. Patent ductus arteriosus, interatrial septal defect, interventricular septal defect and atrioventricularis communis represent the 4 most common lesions. (2) The second subgroup consists of patients with lesions where oxygenated and deoxygenated blood are mixed together, the resultant blood going to both the sytemic and pulmonary circuits. Lesions found are transposition of great vessels, truncus arteriosus, single ventricle; cor biloculare; pulmonic stenosis and nonfunctioning right ventricle, with mitral and aortic insufficiency (Taussig-Bing syndrome); total anomalous pulmonary venous return; and tricuspid atresia with transposition of the great vessels. (3) The third subgroup includes lesions with a right to left shunt of blood on the basis of pulmonary hypertension. The lesions found in this subgroup are reversing patent ductus arteriosus and the Eisenmenger complex.

Fifty per cent of all congenital heart lesions in children is made up by the 4 common left to right shunts. Transposition of the great vessels and truncus arteriosus are the 2 most common admixture lesions.

The roentgenologic evidence of congenital heart disease is largely inferential. The most important facet of this is the pulmonary vasculature. The status of the great vessels may often be gauged by contour as well. The precise delineation of the internal anatomy and physiology of the heart, the great vessels and the other pertinent vessels in congenital heart disease requires certain special procedures.

Retrograde aortography is of great value in extracardiac conditions, particularly in coarctation of the aorta, patent ductus arteriosus, and aortic-pulmonic window.

Angiocardiography is the roentgenographic procedure of choice for definitive diagnosis in patients with right-to-left shunts either on the basis of pulmonic stenosis or pulmonary hypertension and in admixture lesions.

Selective angiocardiography is advocated in certain situations where only one or another feature is in question. For example, the differential diagnosis between valvular and infundibular pulmonary stenosis may be brought out by this method.—*James F. Martin, M.D.*

SCANNELL, J. GORDON, and GRILLO, HERMES C. Primary tumors of the heart; a surgical problem. *J. Thoracic Surg.*, Jan., 1958, *35*, 23–36. (From: The General Surgical Services of the Massachusetts General Hospital and Department of Surgery of the Harvard Medical School, Boston, Mass.)

The authors report 3 cases having had primary tumors of the heart and discuss the characteristic findings and the treatment of these lesions.

The most common primary tumor of the heart is myxoma. The majority of myxomas occur in the left atrium where they arise from the rim of the fossa ovalis in the interatrial septum. They are usually pedunculated and may cause a ball-valve obstruction of the mitral orifice. The result is often a clinical picture indistinguishable in most respects from mitral stenosis. Some clinical features which might suggest intracavitary tumor instead of mitral stenosis are changes in symptoms and murmurs with changing position, a rapid progression of symptoms, and a marked failure of response to medical therapy. Two patients having had myxomas of the heart are described. One had successful removal of an interatrial myxoma and the second died of tumor emboli from an atrial myxoma before the diagnosis could be established. The latter occurrence, namely embolization from atrial myxoma, is sufficiently common to make surgical removal a matter of urgency.

Primary sarcomas, including myxosarcoma, leiomyosarcoma, fibrosarcoma, angiosarcoma, rhabdomyosarcoma, and lymphosarcoma are the second most common primary tumors of the heart. They may occur anywhere in the heart, but are found predominantly on the right side. Hemopericardium is observed frequently. A case of a primary fibrosarcoma of the right atrium is described in detail. The heart was explored because of recurring bloody pericardial fluid which produced tamponade. At exploration, an intramural fibrosarcoma was found and successfully resected.—*Paul C. Olfelt, M.D.*

LIN, T. K., DIEHL, A. M., WALKER, J. W., HALL, T. N., and PEETE, D. C. Unruptured congenital aortic sinus aneurysm with aneurysmal dilatation of the aorta and aortic regurgitation without arachnodactyly. *Am. Heart J.*, March, 1958, *55*, 463–468. (From: The Departments of Internal Medicine and Pediatrics, University of Kansas Medical Center, Kansas City, Kans., and The Children's Mercy Hospital, Kansas City, Mo.)

This report concerns a single case observed and thoroughly studied at the Kansas City Medical Center in 1952. The authors state that the unusual association of an unruptured congenital aortic sinus aneurysm and congenital aneurysmal dilatation of the aorta with aortic regurgitation, but without arachnodactyly or aortic coarctation, has not yet been previously described.

The lesions or anomalies are clearly demonstrated by means of angiocardiography, electrocardiography, and phonocardiography, as well as cardiac catheterization.

The embryologic explanation of these defects is probably based upon a combination of the failure of fusion between the proximal and distal bulbar swellings, and a developmental defect of the elastic tissue at the base of the aorta.

Symptoms of an unruptured aortic sinus, if present, are not specific.

The electrocardiographic changes follow closely the degree of systolic and diastolic overloading caused by these associated anomalies.

Attempts at surgical correction of the aortic sinus aneurysm when unruptured, as in this case, is not justified without the simultaneous alleviation of the severe aortic regurgitation caused by the aneurysmal dilatation of the aorta.—*Lysle Bach, M.D.*

MENGIS, CHRIS L., DUBILIER, WILLIAM, and BARRY, KEVIN G. (New York, N. Y.) The aortic arch syndrome of Takayasu. *Am. Heart J.*, March, 1958, *55*, 435–442.

This syndrome variously called "pulseless disease" or "reverse coarctation" was first described in 1908 by Takayasu. In 1952, a total of 58 cases were collected from the Japanese literature. The usual findings comprise bilateral loss of pulsations in the radial arteries; absence of detectable blood pressure in the arms; ocular changes including the progressive formation of peripapillary arteriovenous anastomoses and cataracts, visual disturbances ranging from photopsia and blurring of vision to blindness; and syncopal attacks frequently associated with convulsions and attributed to hypersensitivity of the carotid sinus. In 1954, a comprehensive review of the literature revealed a total of 28 such cases from the occident. In 36 of 44 Japanese cases in which age and sex were stated, the onset was under the age of twenty-five years, and of these 36 cases, 33 were female. In 15 of the total 28 occidental cases, the onset was likewise under the age of twenty-five, all of them female. In the past two years, 3 more cases of Takayasu's disease have been reported in the occident, and this report deals with another typical case.

The patient is a thirty-five year old married Japanese woman whose chief complaint was "blacking-out" episodes. At the age of fourteen years she was noted, on routine examination, to have no pulses in the upper extremities and apparently very low blood pressure in one arm. At the age of twenty she was

examined for acute trauma to the left eye and told that she had chronic eye disease unrelated to the trauma. At the age of thirty and again at the age of thirty-three she had episodes of transient loss of vision lasting for about fifteen minutes, and began having recurrent occipital headaches which were considered at the time as psychogenic. At the age of thiry-four she experienced a myalgic type of discomfort in the back and right hemithorax which was relieved by change of position. The episode eventuating hospital admission was sudden onset of weakness, nausea, and loss of vision. This episode lasted fifteen minutes, after which she was asymptomatic. She was not wearing constrictive clothing about the neck at the time of the episode. No palpitation, chest discomfort, cyanosis, claudication on mastication, or claudication of the upper extremities had ever been noted.

On physical examination the positive findings were as follows: There was a bruit heard over the right lung posteriorly, along with a palpable thrill over the course of the intercostal arteries in that area. There was a faint pulse in the right radial artery and no pulse in the left. Pulsations of the femoral, dorsalis pedis and posterior tibial arteries were normal. Carotid pulsation was visible on the right side but not on the left. The blood pressure was unobtainable in the left arm, it was 50/40 mm. of mercury in the right arm and 140/70 mm. of mercury in the left leg. The left lens was cataractous and was subluxated into the vitreous; the retina was not visible; there was fine dust-like pigment on the rear surface of the cornea, marked atrophy of the iris, and a fixed irregular pupil 6×5 mm. The right eye was normal.

Serial angiocardiography was done, and demonstrated irregularity in the contour and slight narrowing of the lumen of the aortic arch and descending aorta. There was no opacification of the proximal segments of the innominate, left common carotid, or left subclavian artery. This suggested occlusion of these vessels at their origins. However, with opacification of the descending aorta, large tortuous intercostal arteries were demonstrated along the lower ribs, especially on the right side, where they corresponded to the rib notches and bruits. Subsequent roentgenograms showed reversal of the usual blood flow from the intercostal arteries, through the internal mammary, lateral thoracic and other major vessels of the thoracic wall into the axillary and carotid arteries.

Takayasu's syndrome is ascribed to a degenerative arteritis of the aortic arch and brachiocephalic trunks, with obliteration of the lumina of the latter by an intimal cushion and organized thrombus. Efforts to implicate various infectious, inflammatory, and degenerative processes have thus far been unsuccessful, and the etiology is not apparent. The authors feel that reconstructive vascular surgery offers the only reasonably successful approach to the problem.—*Fred Barlow, M.D.*

Abdomen

Madell, Samuel H. A case of hemangioma (angiomatosis) of the small intestine and mesentery; a radiographic clue to diagnosis. *Radiology*, Oct., 1957, *69*, 564–566. (Address: 2 E. 77th St., New York 21, N. Y.)

The author presents a case report, the purpose of which is to emphasize the diagnostic value of unusual localized clusters of phleboliths in the abdomen as a sign of unsuspected hemangioma of the ileum and mesentery. Because of an unexplained hypochromic anemia and an elevated sedimentation rate, a gastrointestinal evaluation was being undertaken. Barium enema studies showed only a collection of phleboliths in an unusual location suggesting the presence of a vascular tumor. Small bowel examination showed the zone of phleboliths to be encircled by and closely associated with an abnormal loop of proximal ileum which contained many small irregular filling defects and was of irregular caliber. At laparotomy a tumor involving the proximal ileum and its mesentery was found and resected. Pathologic study showed it to consist of tumorous vascular proliferation of both arteries and veins, leading to a classification of angiomatosis.—*Donald N. Dysart, M.D.*

Schmitz-Dräger, H. G., and Thurn, P. Zur röntgenologischen Diagnose und Differentialdiagnose der regionalen Enteritis. (Roentgenologic diagnosis and differential diagnosis of regional enteritis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1957, *87*, 566–574. (From: Medizinische Universitätsklinik, Bonn, Germany.)

There is a steady increase of regional enteritis, an entity which has originally been known as terminal ileitis. Clinical manifestations are not characteristic of the disease, the most constant finding being either simple or bloody diarrhea. Only on the basis of roentgen studies can a definite diagnosis be established. Barium enema examination is most helpful when the lesion is limited to the terminal ileum. Since, however, multiple lesions are not infrequently observed from the stomach down to the colon, oral barium study is indicated in all cases.

Four representative cases are reported and illustrated, all of which have been surgically verified. The first was a male, age twenty-three, with complaints of bloody diarrhea, periumbilical pain and weight loss for the past six months. Barium enema study revealed a moderate regional enteritis of the terminal ileum. The second case was also a male, age twenty-two, with a thirty months' history of bloody diarrhea and periumbilical pain. Appendectomy had been performed two years previously at which time the diagnosis of regional enteritis had been established. There were anal fissures and mod-

erate malnutrition. Roentgen study revealed a more advanced involvement of the terminal ileum with adhesions to the sigmoid colon. In the third case, a male age twenty-two, there was a history of bloody diarrhea with cramps and weight loss for the past five years. The diagnosis of regional enteritis had also been established during an appendectomy four months prior to admission. This was followed by an operation for an abscess formation. There was a palpable ileocecal tumor with subfebrile temperature. Roentgen study revealed regional enteritis of the entire ileum; stenotic segments alternated with areas of dilatation. Wide separation of small intestinal loops, so-called stenosis peristalsis, effacement of mucosal pattern and a small fistula leading to the anterior abdominal wall were noted. In the fourth case, a male age twenty-two, appendectomy with resection of an inflammatory tumor had been performed eight years previously. Watery diarrhea and weakness, however, had persisted. Roentgen study revealed duodenal involvement with mucosal thickening and stenosis. Similar lesions were also noted lower down in 3 separate segments of the small intestine. The intervening segments or skipped areas were intact.

The typical roentgenologic changes were: (1) Involvement of the wall of the intestine with thickening, rigidity, separation of loops and stenosis; (2) mucosal alteration with coarsening of loops, polypoid and reticular changes or effacement of the pattern; and (3) secondary changes consisting of adhesions, internal and external fistulae and abscess formations. From a surgical standpoint it is important to differentiate between the stenosing and the nonstenosing form.

In the differential diagnosis chronic inflammatory changes, deficiency patterns and tumors have to be excluded. When the lesion is localized in the ileocecal area, differentiation from tuberculous involvement and from ulcerative colitis may occasionally be difficult by roentgen studies alone.—*Ernest Kraft, M.D.*

Clatworthy, H. William, Jr., and Lloyd, James R. Intestinal obstruction of congenital origin; a study of diagnosis and management on one hundred sixty-three cases. *A.M.A. Arch. Surg.*, Dec., 1957, *75*, 880–890. (Address: H. W. Clatworthy, Jr., 695 Bryden Rd., Columbus, Ohio.)

One hundred sixty-three consecutive cases of intestinal obstruction of congenital origin admitted to Children's Hospital, Columbus, Ohio, were reviewed in order to assess the factors contributing to death in fatal cases. In retrospect, 43 of the 56 infant deaths were considered possibly preventable. The causes of death were diagnostic inaccuracies in 22, technical errors in surgery and management in 12, infection in 9. The unavoidable causes were associated anomalies, mucoviscidosis, and inadequate intestine.

Since over half of avoidable deaths were the result of incomplete or late diagnosis, particular attention was given to the signs and symptoms which might have led to earlier diagnosis. The four cardinal manifestations were abdominal distention present in 129, bilious vomiting in 83, abnormality of meconium evacuation in 147, and maternal polyhydramnios in 17 cases.

The cases were grouped according to six types of intestinal obstruction.

Intrinsic obstruction (atresia or stenosis) occurred in 48 cases. In high obstruction, widely dilated duodenums should be opened to avoid missing an intrinsic web. In obstructions of lower jejunum and ileum, it may be advisable to do very extensive resections, including the markedly dilated proximal intestine and the microsized distal intestine. Experience indicates that infants tolerate extensive resection of small intestine well, but prolonged careful hospital management is required while the adaptation of the remaining small bowel takes place. The over-all mortality in this group was 45 per cent.

Neuromuscular defects were responsible for 46 of the obstructions, and in this group there were 10 deaths, all but two with very extensive involvement were possibly preventable. Four patients died of progressive obstruction or perforation while on medical therapy; three were incorrectly diagnosed and not treated; and one, a six month old infant, died of pelvic peritonitis following a primary pull-through procedure. A temporary loop colostomy in the sigmoid area is recommended in preference to early definitive surgery. The pull-through procedure is now delayed until eighteen to twenty months of age.

Anomalies of rotation and fixation accounted for 14 deaths, 11 resulting from failure to suspect the diagnosis until the entire midintestine was nonviable. In 3 cases intrinsic duodenal obstructions were missed at the time the Ladd procedure was performed.

Intraluminal obstruction by an obturating mass of viscid meconium accounted for 28 obstructions. Twelve of these were in mid or terminal ileum and subsequent studies showed evidence of pancreatic fibrosis. All of these have succumbed to or suffer from respiratory complications of pancreatic fibrosis.

In 14 cases the obstructive mass was in the colon, and although initially there was an absence of enzymal gelatinase in the meconium, there have been none of the other complications of pancreatic fibrosis. All were well up to six years later.

Extrinsic obstruction (4 cases) was present in 4 cases, 2 of which were due to annular pancreas.

Meconium peritonitis may complicate other forms of obstruction. All in this group succumbed.—*Lois Cowan Collins, M.D.*

Levene, George. Rates of venous absorption of carbon dioxide and air used in double-contrast examination of the colon. *Radiology*, Oct., 1957, *69*, 571–575. (Address: Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Mass.)

The author compared carbon dioxide with air as a medium for double-contrast study of the colon. The carbon dioxide was found to have been completely absorbed in forty-five minutes whereas the air was still present and causing discomfort after two hours.

The author concludes that carbon dioxide as a contrast medium for roentgenologic examination of the colon has the following advantages: (1) It is rapidly absorbed; (2) it readily goes into solution in the blood stream, thereby eliminating the danger of embolus; and (3) in case of bowel perforation, it is quickly absorbed from the peritoneal cavity.—*John Alexander, M.D.*

Leupold. F., and Heuck, F. Untersuchungen über die Ausscheidung des Gallekontrastmittels "Biligrafin" bei Gesunden und Kranken. (Studies of the excretion of the gallbladder contrast medium "biligrafin" in the healthy and in the diseased.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1957, *87*, 443–451. (From: Medizinische Universitätsklinik, Kiel, Germany.)

The authors studied the blood serum level and urine excretion of biligrafin in normal and diseased individuals, in order to clarify the factors which influence its excretion and to establish if the determination of the biligrafin, namely iodine, excreted in the urine actually serves as a liver function test. Since the examination is not entirely free of danger, the material was restricted to a small number of patients. In normal subjects there is a rapid fall in the serum iodine during the first hour after the injection. Urinary excretion of the iodine varies widely even in normal individuals.

The results of the study may be summarized as follows: A delay in the fall of serum biligrafin levels may be associated with increased iodine excretion in the urine, as in biliary stasis and circulatory decompensation, or with decreased iodine secretion in the urine, as in increased diuresis in thirsty patients and in renal insufficiency.

The authors agree for the most part with Hornykierwytsch that "a negative cholangiogram is a reliable sign of severe liver damage; a positive cholangiogram, however, does not exclude liver disease." Certain exceptions do occur.

The most important factors which influence the excretion of biligrafin through the bile and urine are: (1) The amount of the injected contrast medium; (2) size and weight (plasma volume) of the patient examined; (3) status of liver parenchyma; (4) status of renal parenchyma; (5) status of circulatory system; (6) decrease in diuresis; (7) protein combination and plasma albumin spectrum; (8) amount of deposited fat present (?); and (9) tubular re-absorption (?). These various factors may overlap or add to one another. Parenchymal liver damage, as long as there is no marked bile stasis, is no contraindication to a biligrafin study. This study is not indicated in acute liver damage or in circulatory decompensation. An absolute contraindication, besides sensitivity to the contrast substance, is the presence of both liver and kidney damage.—*Samuel Richman, M.D.*

Ramos, Albano, de Carvalho, J. Rodrigues, Rosa, Freitas, and Fernandes, Machado. Un aspecto raro de colangiograma intravenosa. (A rare aspect of intravenous cholangiography.) *Acta iber. radiol. cancerol.*, Jan–March, 1957, *12*, 1–5. (Address: Praça Guiherme Gomes Fernandes, 92. Porto, Portugal.)

An unusual demonstration of Ascaris lumbricoides in the common bile duct by means of intravenous cholangiography is reported.

A patient, seventy-three years of age, had suffered attacks of colic in the right upper abdominal quadrant for fifteen years. For the past three months before admission these attacks had increased in severity and frequency. Oral administration of telepaque revealed a gallbladder of moderately dense opacification. Intravenous biligrafin study showed a long filling defect in the opacified common duct. The history disclosed that the patient suffered from ascariasis and had eliminated the parasite in the stool.

At operation it was found that the Ascaris had been evacuated from the common duct. Cholecystectomy was performed with a resultant histologic diagnosis of cholecystitis.—*Charles M. Nice, Jr., M.D., Ph.D.*

Pohlandt, Kl. Cholezystographische Diagnose gutartiger Gallenblasentumoren (Papillome). (Cholecystographic diagnosis of benign tumors [papilloma] of the gallbladder.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1957, *87*, 451–465. (Address: Kl. Pohlandt, Am Dobben 105, Bremen, Germany.)

The author reviewed the literature regarding papilloma of the gallbladder and noted that the condition has been found much more frequently in the United States than in Europe. He believes that about one per cent of papillomas is found in gallbladders which have been removed. These tumors are 1 to 5 mm. in size, although some as large as

1.5 cm. have been reported. They are usually multiple in number and according to one report are associated with gallstones in 68 per cent of cases. Kirklin reported one carcinoma in 1,400 papillomas. The author believes that papilloma is not the result of lithiasis.

The cholecystographic basis for the diagnosis of papilloma rests on the demonstration of filling defects, single or multiple. An extremely important factor is a careful roentgenographic study of the gallbladder in the contracted state after a fatty meal, since these defects show up much more clearly at that time. An adenoma causes only one defect, somewhat larger and nearly always in the fundus of the gallbladder. Change in position of the defects suggests gallstones. Six cases of papilloma of the gallbladder are presented, none in combination with gallstones. In 3 of the cases, operative proof was obtained.

The author emphasizes that the excised gallbladder must be handled carefully so that a pedunculated papilloma may not be inadvertently crushed or pushed out. Some very convincing roentgenograms are reproduced.—*Samuel Richman, M.D.*

Martinez, Nestor S., Morlock, Carl G., Dockerty, Malcolm B., Waugh, John M., and Weber, Harry M. Heterotopic pancreatic tissue involving the stomach. *Ann. Surg.*, Jan., 1958, *147*, 1–12. (From The Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Heterotopic pancreatic tissue is defined as pancreatic tissue outside the boundaries of the pancreatic gland. The article is confined to heterotopic pancreatic tissue limited to the gastric wall. The authors have selected 51 surgical cases from a group of 68 cases recorded at the Mayo Clinic between 1907 and 1954. They divide the 51 cases into those with clinically significant findings and those in which the finding is thought to be incidental. The condition is conceded to have no specific symptom complex. Occasionally, pyloric obstruction is produced by prolapse of the lesion through the pylorus, and severe gastrointestinal bleeding has occurred because of ulceration of the overlying gastric mucosa. Roentgenologic demonstration of these lesions justifies surgical excision and confirmation of its character.—*Richard E. Kinzer, M.D.*

Shapiro, Robert. Experimental opacification of the pancreas; a preliminary report. *Radiology*, Nov., 1957, *69*, 690–692. (Address: Hospital of St. Raphael, 1450 Chapel Street, New Haven 11, Conn.)

In this paper the author gives a preliminary report of experimental studies pertaining to contrast visualization of the pancreas. Recent experiments have shown that the pancreas is concerned with the metabolism of zinc. In cats the average concentration of zinc was 7 times greater in the fibrotic pancreas than in normal control animals. Intravenous injection of radioactive zinc showed that a large proportion of this labeled zinc was excreted in the pancreatic juice and very little appeared in the bile. These findings indicate that zinc not only is excreted in normal pancreatic juice but, more important, is actually concentrated by the acinar cells.

With this background the theoretical application for contrast roentgenography of the pancreas is relatively clear, *i.e.*, the preparation of a haloginated, soluble, and nontoxic zinc compound, in which the halogen acts to produce radiopacity, and the zincation to direct the compound to the pancreas. Several such compounds have been tried, but attempts to overcome the heavy metal (zinc) toxicity have as yet been unsuccessful.—*Donald N. Dysart, M.D.*

Gynecology and Obstetrics

Regidor, Peña. Die Vor- und Nachteile der öligen und wasserlöslichen Kontrastmittel in der Salpingographie. (Advantages and disadvantages of oily and water-soluble contrast media in salpingography.) *Röntgen Blätter*, Dec., 1957, *12*, 380–384. (From: Hospital de los Desemparados, Santa Cruz de Tenerife, Canary Islands, Spain.)

The author tried endografin 50 per cent [Schering, Berlin] which is an aqueous contrast medium in 25 salpingographies. He found that this contrast medium passes rapidly through the mural part of the tube and enters the peritoneal cavity if the tubes are open. This rapid passage has the disadvantage that details of uterus and tubes are not visualized on the roentgenograms and that the roentgenograms show other densities caused by the presence of contrast medium in the peritoneal cavity. The advantage of endografin and other aqueous contrast media is the immediate disclosure of the patency of the tubes which in many cases is the reason for the salpingography.

On the other hand, oily contrast media possess the advantage of visualizing all details on the roentgenogram. They show the structure of the uterine mucosa and can reveal endometriosis. A disadvantage is the occurrence of so-called "false occlusions" which are caused by the high viscosity of the oily contrast medium or by spastic contraction of the tubes. To avoid the latter, application of an oily contrast medium should always be preceded by injection of an antispasmodic or a drug which blocks the ganglia of the vagus (buscopan) and followed by an air-insufflation in order to convey the remaining oil from the cavity of the uterus to the abdominal cavity.—*Hans Lewin, M.D.*

Erbslöh, Joachim. Über die Vor- und Nachteile wasserlöslicher Kontrastmittel. (Advantages and disadvantages of water-soluble contrast media.) *Röntgen Blätter*, Dec., 1957, *12*, 384–389. (From: Geburtsh.-Gynäk. Abteilung des Kreiskrankenhauses Stormarn, Bad Oldesloe, Germany.)

This publication refers to the foregoing paper of Peña Regidor on the same subject.

The author reports his results based on 250 salpingographies with the water-soluble contrast medium endografin which is chemically identical with biligrafin [in U.S.A. under the name of cholografin] used for intravenous cholangiography. The disadvantages of oily contrast media are late side effects as oil cysts, granulomata and chronic inflammatory reactions, but they are well tolerated during the injection.

Water-soluble contrast media are completely absorbed within a few hours and not later than a few days, and do not produce late side effects. One disadvantage of all aqueous contrast media is an early reaction two or three days after the examination, namely, a reactive hyperemia and an exudation of the visceral and parietal peritoneum which later disappears completely but makes necessary a complete bed rest for twenty-four hours after injection. Other disadvantages of aqueous solutions are the following: (1) the injection is painful necessitating an anesthetic, although endografin proved to be painless; (2) the addition of a "viscosity factor" in order to increase the viscosity makes it nearly impossible to press the contrast medium through the intramural portion of the tube which has a lumen of only 0.8 mm. Some of these viscosity factors produce granulomata in the myometrium, the tubes, ovaries and peritoneum. Some of them are carcinogenic. The great advantage of endografin is its high viscosity which approximates that of 40 per cent iodipin. Endografin shows very well all details on fluoroscopy. Re-examination has to be done after a few minutes up to one and one-half hours, not after twenty-four hours as with oily contrast media. There are rare exceptions. One of these is a case, observed by the author, where the endografin was still visualized after twenty-four hours in a hydrosalpinx.

To avoid possible spasm of the tubes, the author regularly injects 0.02 gm. morphine and 0.0004 gm. scopolamine one hour before the salpingography.

Late reactions with aqueous solutions are exceptions but do occur. The author observed in 250 salpingographies during a period of three years only five times a slight rise of temperature up to 37.3° C. for a short time and only three times a more serious late reaction.—*Hans Lewin, M.D.*

Lincoln, Thomas A., and Gupton, Edwin D. Radiation dose to gonads from diagnostic x-ray exposure. *J.A.M.A.*, Jan., 1958, *166*, 233–239. (Address: T. A. Lincoln, Post Office Box P, Oak Ridge, Tenn.)

A method for the measurement of radiation to the gonads is described. The amount of radiation during various diagnostic examinations was measured. Correlation of these data with the number of roentgen examinations carried out on employees of the Oak Ridge National Laboratories resulted in an average total gonadal dose per year of 0.013 rads for males and 0.0351 rads for females. The importance of minimizing the number of abdominal examinations is emphasized. Physical factors that will decrease the amount of radiation to the gonads are discussed.—*Jack Purcell, M.D.*

Skeletal System

Donnan, M. G. F. (Perth, W. Australia). Fluorosis. *Proc. Coll. Radiologists, Australasia*, June, 1957, *1*, 1–2.

Fluorosis is a disease characterized by nondestructive generalized osteopetrosis of the skeleton associated with dental mottling and spicular bone deposits at the site of muscle insertions and ligamentous attachments, leading to multiple stiff points and a "poker" spine. It is caused by drinking water that contains fluorine in excess of one part per million over long periods.

Fluorosis has been reported as occurring in animals and man in practically every country in the world.

The only foods rich in fluorine are sea-foods and tea.

Fluorine is well known as an enzyme inhibitor. The kidneys seem to show the greatest sensitivity to toxic levels of fluorine with resultant cytologic and structural change. In addition, it has an inhibitory effect on gastric acid secretion. Many people exposed to fluorine in the cryolite industry commonly complain of gastric symptoms.

The effect of fluorine intoxication appears to vary with the age of the person and the dose of the mineral. In young persons the bones may show excessive osteoporosis, whereas in older persons the bones become dense and sclerotic with surrounding spicule formation.

The interpretation of pathologic changes is made more difficult by the frequent association of renal damage, metastatic calcification and hyperplasia of the parathyroid glands. The urine contains albumin and fluorine in amounts far in excess of the normal upper limits.

Albers-Schönberg's disease, sclerosing carcinomatous metastases, osteitis deformans and osteomyelosclerosis bear a superficial roentgenographic resemblance.

The roentgen appearance may be summarized as "a solid candy skeleton with fluffy icing at the margins."

The disease in itself does not appear to affect the

intervertebral disk or the cartilage of joints. A conspicuous feature is that the changes become less discernible peripherally.—*Stephen N. Tager, M.D.*

Oliva, Luigi, and Farris, Guido. Endostosi e periostosi leprose. (Endosteal and periosteal changes in leprosy.) *Radiol. med.*, Dec., 1957, *43*, 1174–1195. (From: Istituto di Radiologia e Clinica Dermatologica, Università di Genova, Genova, Italy.)

The authors have observed in the years 1935 to 1956, 54 cases of leprosy, the majority of which presented also skeletal changes.

The lesions are divided as follows: (A) specific osteopathies: (1) osteoplastic lesions (periostitis, osteomyelitis), (2) osteolytic lesions (cystic areas of rarefaction, etc.); (B) osteopathies due to neurotrophic disturbances (absorption of bone with amputation of the terminal phalanges, fractures, etc.); (C) arthropathies: (1) specific, (2) neurotrophic.

The site of involvement was in the tibia and fibula in 25 patients; in the fibula only, in 11; in the femur in 2; in the lumbar spine in 12; in the ulna and radius in 10. It was possible to recheck after three years only 6 patients with lesions in the tibia and fibula, and 3 with lesions in the radius.

The most frequent leprous changes observed in the fibula and tibia were characterized by periosteal reactions, circumscribed or diffused, and of various degrees of severity in accordance with the stage of the lesion. In the radius and ulna the most common bony changes were due to endosteal reaction, with thickening of the cortex, narrowing of the medullary canal, etc. The alterations affecting the lumbar spine were of the spondyloarthrotic type. Of interest was the observation that there was no correspondence between the nodular form of leprosy and the productive changes in the bones, or between the neural form of leprosy and the trophic bony alterations. In fact, the endosteal and/or periosteal lesions were more prominent in the neural leprosy.

In discussing the various underlying causes of the bony changes in leprosy, the authors mention that they have noted, in patients with lesions localized in the lower extremities, an altered circulation, with stasis, trophic ulcers and deposits of hemosiderin,—all changes which are most commonly seen in patients with venous stasis of the legs and especially with varices.

This observation, in their opinion, could explain the endo- and periosteal changes on the basis of altered venous circulation producing trophic disturbances, modifications of the calcium and phosphorus balance, and eventually the productive changes in the bones.—*A. F. Govoni, M.D.*

Wieser, C., and Isler, U. M. Viszerale Röntgenbefunde bei Osteomyelosklerose. (Visceral roentgen findings in osteomyelosclerosis.) *Radiol. clin.*, Nov., 1957, *26*, 329–333. (From: Zentrales Röntgeninstitut und Med. Klinik des Kantonsspitals, St. Gallen, Switzerland.)

Osteomyelosclerosis is characterized by splenomegaly, leukoerythroblastosis and bone marrow fibrosis. Further advanced cases may show osteosclerosis, visualized on roentgenograms. Splenomegaly represents a tumor-like process of the reticulohistiocytic system (neoplastic reticulosis). This process need not be limited to the spleen but may also involve other organs where true tumors may develop. Therefore, in addition to the bone changes, even vague visceral roentgen findings may be significant in osteomyelosclerosis. This is illustrated by the following two cases.

The first case is that of a fifty-eight year old male patient with a history of proved osteomyelosclerosis of five years' duration. Roentgenograms of the skeleton showed typical diffuse, partially patchy osteosclerosis, diminishing toward the periphery. Retrograde pyelography revealed hydronephrosis of the left kidney. Two years later the patient died of a traumatic brain hemorrhage. Autopsy findings: osteomyelosclerosis; hepatosplenomegaly; tumor-like masses of marrow in the spleen, in both kidney hili and subpleurally; stenosis of the right upper ureter due to tumor masses; obstruction of the left ureter by a urinary calculus; bilateral hydronephrosis. Significant were the peripelvic tumors of the kidneys representing extraosseal blood forming centers in neoplastic reticulosis.

Formation of urinary calculi and tumors of the kidney hili with secondary hydronephrosis are typical of osteomyelosclerosis.

The second case is that of a sixty-three year old male patient. Fluoroscopic and roentgenographic examination of the colon revealed a tumor which was confirmed on three examinations following at short intervals. Roentgenograms of the spine showed marked osteosclerosis of the lumbar vertebrae. There was marked splenomegaly. The laboratory findings disclosed a normochromic anemia with 47 per cent Hb, 4,400 leukocytes and leukoerythroblastosis. Bone biopsy demonstrated typical osteomyelosclerosis. Following numerous blood transfusions there was general improvement and also improvement of the anemia. Four months later the cecal tumor could be visualized on roentgen examination.

Cases in the literature as well as the authors' second case prove the occurrence of neoplasms of the intestinal tract in patients with osteomyelosclerosis.

The authors cannot explain the disappearance of the cecal tumor. However, tumor regression has been reported in other neoplastic reticuloses, especially Hodgkin's disease, which show some general similarity with osteomyelosclerosis.—*Hans Lewin, M.D.*

Gilmer, W. Scott, Jr., and MacEwen, G. Dean. Central (medullary) fibrosarcoma of bone. *J. Bone & Joint Surg.*, Jan., 1958, *40-A*, 121–141. (From: The Campbell Clinic and the Department of Pathology and Microbiology, University of Tennessee Medical Units, Memphis, Tenn.)

Malignant tumors involving bone recorded at the Campbell Clinic from 1930 through 1955 totaled 227. The authors identified 9.7 per cent of these as central or medullary fibrosarcomas.

Review of the literature failed to disclose any conclusions concerning the prognostic implication of central fibrosarcoma and the purpose of this study was to further define this type of tumor as a separate entity and record experience with it.

A series of 22 cases is presented and a table lists each case as to age, sex, symptoms, duration of symptoms, location of lesion, grade, treatment, and follow up data. Six reproductions of roentgenograms are included, some as negatives and some as positives. It is difficult to visualize the tumors from the illustrations presented. Photomicrographs and photographs of hemisections of gross specimens are also shown.

The authors conclude that central or medullary fibrosarcoma is an entity. They point out that the five year survival rate is much worse than in soft tissue sarcoma, but better than in true osteogenic sarcoma.—*Richard E. Kinzer, M.D.*

Aronstram, Elmore M., Ludden, Ted E., and Matuska, Walter H. Chondrosarcomas of the thoracic spine; report of two cases. *J. Thoracic Surg.*, 1956, *31*, 725–730. (From: The Thoracic Surgery Service, Pathology Service, and the Surgical Service, Valley Forge Army Hospital, Phoenixville, Pa.)

Chondromatous tumors of the thoracic spine presented as posterior mediastinal masses in an asymptomatic thirty-two year old male and associated with pleuritic pain and sanguineous effusion in a twenty-year old female are described. The lesions were removed without incident from their attachments to mid-dorsal vertebrae and each satisfied gross and histologic criteria for chondrosarcoma.

No previous reports of verified primary cartilaginous malignancies in this area were found.—*Dale Undem, M.D.*

Newton, Thomas Hans. Cervical intervertebral-disc calcification in children. *J. Bone & Joint Surg.*, Jan., 1958, *40-A*, 107–113. (From: The Department of Radiology, Children's Medical Center, Boston, Mass.)

Seven children with calcified cervical intervertebral disks are reported and the roentgenograms of 4 of them are reproduced. The author points out that intervertebral disk calcification is a common degenerative process in later life. He notes that it has rarely been reported in children and he states that calcified cervical disks in childhood are usually associated with symptoms in direct contradistinction to the calcified disks found in later life which are usually asymptomatic. Most of the patients recovered rapidly from their symptoms and their serial roentgenograms showed that the disk calcification gradually disappeared.

It is emphasized that there is much similarity between this type of calcification and calcific tendinitis. The author believes that this syndrome is probably much more common than reports in the literature would suggest.—*Richard E. Kinzer, M.D.*

Blood and Lymph System

Decker, K. Befunde am Halsteil der A. carotis interna im Angiogramm. (Findings in the cervical portion of the internal carotid artery in angiography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Dec., 1957, *87*, 693–707. (From: Nervenklinik der Universität, München, Germany.)

Since signs of cerebral disease may be due to pathologic changes in the cervical portion of the carotid arteries, cervical angiograms deserve careful study, especially if a diagnosis cannot be reached by cerebral angiography. The radiologist should be familiar with certain normal variants pertaining to the cervical portion of the internal carotid arteries. It is important to remember that in infants the bifurcation of the common carotid artery is at the level of the angle of the jaw, whereas in adults it may be at the level of the sixth cervical vertebral body. The lower the bifurcation, the more difficult it is to inject the internal carotid artery at its site of origin. Furthermore, in such instances the internal carotid artery is often thinner than normal. Thus a narrow caliber does not always denote a pathologic change. Other normal variants include tortuosities and bucklings of the cervical portion of the carotid arteries. It is interesting to note that these variants are often associated with some dilatation of the vessels.

Certain changes occur as a result of the needle puncture or the injection of contrast media. A rapid injection may cause localized circular contractions of the vessel. Sometimes the needle puncture alone produces a vascular contraction. Similar changes may result as a chemical response or even as a physical response to a cold solution. As a rule the patients are unaware of these changes. However, the procedure is not altogether harmless because of the following types of possible complications: (1) Subintimal hemorrhages are not always avoidable and may lead to immediate or delayed local or cerebral damage; (2) even physiologic saline solution may cause damage to the vascular wall at the site of the injection; (3)

injection of 1 or 2 cc. of contrast material into the intima may cause a complete occlusion of the vessel. Even though the contrast material is absorbed fairly rapidly, the indentations in the vascular lumen may persist for a long time. If thrombosis ensues, none of the contrast material will enter the intracranial circulation, but the carotid artery may be opacified from the site of injection up to the level of the ophthalmic artery. However, most of the changes in the cervical portion of the carotid arteries are usually reversible.

The differential diagnostic features of cervical angiography include the following: (a) Localized, well circumscribed lesions are usually due to the needle puncture, especially if they coincide with the site of puncture. Intimal hemorrhage occurs at the site of injection or on the posterior wall and often cannot be differentiated from spastic contractions; the same may be said for intramural injections of saline solution. Contrast material injected into the wall of the vessel is easily recognized. Arteriosclerotic changes are often multiple and are associated with circular narrowings, buckling, or aneurysmal dilatations; (b) widespread narrowing as a result of the injection or merely the needle puncture may resemble a string of pearls. Such a change is usually confined to the cervical region and has not been observed beyond the carotid syphon. Vascular contraction following intracerebral hemorrhage has not been demonstrated proximal to the syphon. Re-examination after a previous angiographic study may disclose a long stretch of smoothly narrowed vessel without any dent to mark the site of the previous injection. Nasopharyngeal tumors may cause an indentation of the carotid artery without displacement; complete occlusion is rare; displacement of the vessel and pathologic vascularization of the tumor are of course easily diagnostic. Hypoplasia of the cervical portion of the carotid artery is rare but in such cases, the vessel is uniformly thin in its entire cervical course. In one patient, the internal carotid artery ended as the ophthalmic artery, whereas the area supplied by the anterior and middle cerebral arteries was fed from the contralateral internal carotid artery. An embolus or contraction of the common carotid artery cannot be identified by cervical angiography. Arteriosclerosis cannot be differentiated from Buerger's disease, although irregular and poorly circumscribed narrowings favor the latter diagnosis. Two patients with luetic endarteritis showed a generalized narrowing up to the carotid syphon.—*J. Zausner, M.D.*

PASTOR, BERNARD H., and BLUMBERG, BERNARD I. Persistent left superior vena cava demonstrated by angiocardiography. *Am. Heart J.*, Jan., 1958, *55*, 120–125. (From: The Medical Service, Veterans Administration Hospital, Philadelphia, Pa.)

The authors have reviewed the literature on anomalies of the superior vena cava and present a case of persistent left superior vena cava demonstrated by angiocardiography. The varieties of persistent left superior vena cave were classified by McCotter in 1916 in a detailed study of 120 cases. Winter in 1954 classified the variations in reviewing 174 cases in more detail.

The most common variety of persistent left superior vena cava drains into the coronary sinus. The authors discuss the embryologic development of the vena caval system and it is pointed out that failure of development of the anastomosis between the inferior cardinal veins is considered responsible for persistence of the left superior vena cava.

Prior to the advent of cardiac catheterization and angiocardiography, vena caval anomalies were rarely diagnosed clinically. The first demonstration of a left superior vena cava during life is credited to Castellanos in 1944. Since that time several observers have noted such anomalies during catheterization or angiocardiography. When the catheter is advanced from the left arm and enters the heart in an abnormal position, an anomalous left superior vena cava may be suspected, while from the right arm the catheter may enter a left superior vena cava by way of the coronary sinus.

Normally, angiocardiography readily discloses a persistent left superior vena cava if the left arm is injected. However, angiocardiography performed from the right side or via the femoral vein may not visualize a left superior vena cava unless there is a large anastomosis.

A left superior vena cava entering the right atrium may produce no physiologic disturbance although it may interfere with cardiac catheterization, provide obstruction at the time of surgery or be important in calling attention to associate anomalies.

If the left superior vena cava enters the left atrium, it constitutes a serious malformation, particularly if the right superior vena cava is absent. Fortunately this is an extremely rare anomaly.

The authors present a case of an anomalous left superior vena cava discovered in a patient with clinical aortic stenosis. Chest fluoroscopy, however, revealed a large left atrium in addition to enlargement of the left ventricle. In order to determine the possibility of a concomitant mitral stenosis, angiocardiography via the left arm was performed. This disclosed an anomalous venous channel which entered the right atrium by way of the coronary sinus. Because this injection did not permit visualization of the left cardiac chambers, a second study ulilizing the right arm was performed which showed a normally situated superior vena cava.—*William C. MacCarty, Jr., M.D.*

JOHNSTON, E. D. (Townsville, Queensland, Australia). Percutaneous splenic portography

or splenic portal venography. *Proc. Coll. Radiologists, Australasia*, June, 1957, *1*, 3–9.

Splenic portography has developed mainly due to progress and improvement in vascular surgery, in which various anastomoses between the portal and systemic venous systems for the relief of portal hypertension have been developed. The author reviews the pathologic background and the indications for the procedure.

The operations designed to prevent further bleeding from esophageal varices may be divided into those aimed at relieving portal hypertension and those aimed at removing the varicose area. Their particular indications depend upon several factors of which the most important are (a) the cause of the portal hypertension, (b) the site of obstruction to the blood flow, and (c) the anatomy of the portal venous system.

The indications for splenic portography are, in the order of importance: (1) To provide information for the surgeon in order to assist him in the planning of a surgical attack on the portal hypertension; (2) to assist in the discovery of the cause of portal hypertension and to assist in the diagnosis of hepatic and splenic enlargement; (3) in the investigation of obscure upper abdominal masses and in the case of radical surgery to indicate whether the portal vein is involved in the malignant mass.

The technique is described, in detail.

Complications may arise: (1) Due to iodine sensitivity; (2) due to unsuccessful splenic injection. The opaque medium may be injected into: (a) the peritoneal cavity, (b) the colon, and (c) the stomach; (3) the splenic complications are the important ones. The most frequent one is due to retrograde spread along the needle tract. This is not uncommon and is presumably due to the puncture being rather shallow.

The alterations in the portal system will naturally depend on the obstruction, the degree and the type of obstruction, the degree and the type of collateral circulation.

First, the obstruction is either intrahepatic or extrahepatic. In the intrahepatic block, filling of the splenic and portal veins and the intrahepatic vessels is observed.

Other collateral systems are frequently evident. Short gastric veins are seen connecting the splenic vein with the fundic plexus; collateral veins are also seen over the spleen connecting with the intercostal veins and systemic retroperitoneal veins and over the anterior abdominal wall. On the right side a few collateral veins may be seen over the upper border of the liver.

The second diagnostic feature is retrograde flow into the superior and inferior mesenteric veins. Flow into the inferior mesenteric vein is the one commonly seen.

In the extrahepatic block the site of the obstruction in the portal vein or the splenic vein can often be visualized.

Portal block is demonstrated by the absence of filling of the intrahepatic veins, or delayed filling with poor concentration of the opaque medium; in the presence of a collateral circulation and in the absence of a normally filled portal vein.

Splenic vein obstruction can be clearly shown if it is sited hepato-proximally, the vein being filled to some extent, with a collateral circulation evident via the gastric veins and between the spleen and anteroposterior abdominal wall and the intercostal veins.

The splenic obstruction is usally due to a thrombosis, but may be due to a pancreatic mass or enlarged lymph nodes.

When the obstruction in the splenic vein is hepatodistal, diagnosis is not so easy.

Absence of filling of the splenic vein and the presence of a collateral circulation favor the diagnosis of splenic vein obstruction.

In cirrhosis it is said there is a characteristic pattern for the intrahepatic vessels; the vessels being finer and less numerous.

The examination has been used also in cases of hepatic tumor and liver enlargement of unknown nature, and in pancreatic tumors and retroperitoneal masses. In the liver, it may show the intrahepatic veins displaced or curved, apparently surrounding a hepatic mass. In the pancreas, a mass may be demonstrated by displacement of the veins.—*Stephen N. Tager, M.D.*

Cassel, William G., Spittel, John A., Jr., Ellis, F. Henry, Jr., and Bruwer, André J. Arteriovenous fistula of the splenic vessels producing ascites. *Circulation*, Dec., 1957, *16*, 1077–1083. (From: The Mayo Clinic and the Mayo Foundation, Rochester, Minn.)

Ascites may result from a splenic arteriovenous fistula, either because of resultant portal hypertension, or hepatic congestion, or a combination of both.

The successful cure of ascites produced by an arteriovenous fistula of the splenic vessels is reported. A thirty-four year old woman developed ascites, peripheral edema, fever and dyspnea about six weeks after termination of a normal pregnancy. History disclosed that she had sustained a bullet wound in the left upper abdomen eleven years earlier. Physical examination revealed evidence of ascites, enlargement of liver, and a continuous multiple pitched murmur over the left posterolateral part of the thorax about 2 cm. lateral to the bullet scar. Roentgenograms demonstrated calcified cyst-like structures in the left upper quadrant, lateral to kidney. Aortography showed opacification of the area within the calcification (splenic artery aneurysm) and a later phase exposure showed tremendous dila-

tation of the splenic vein, a little below and medial to the arterial aneurysm.

Both the arterial and venous aneurysm, the spleen, and a portion of the tail of the pancreas were removed. A 4 mm. communication between the arterial aneurysm and venous aneurysm was found. The patient made an uneventful recovery and three months postoperatively was well and had noted no further ascites.

It seemed possible that this arteriovenous fistula might have been the result of either the earlier trauma, or perhaps rupture of a previously existing aneurysm of splenic artery into the splenic vein as a result of changes in intra-abdominal pressure incident to parturition.

Five previously reported cases are cited, in only one of which had there been ascites.—*Lois Cowan Collins, M.D.*

Abrams, Herbert L. The vertebral and azygos venous systems, and some variations in systemic venous return. *Radiology*, Oct., 1957, *69*, 508–526. (Address: Stanford University School of Medicine, San Francisco 15, Calif.)

The systemic venous drainage of man returns to the heart through 2 great vessels, the superior and the inferior vena cava. An effective communication between the 2 caval systems is accomplished by the vertebral veins and the azygos system. This is made possible partly through the rich communications of the vertebral veins with the veins of the neck, thorax, abdomen and pelvis. Although their presence has been well known for over a century, Batson revived interest in the vertebral system of veins by demonstrating that a thin opaque medium injected into the dorsal vein of the penis of a cadaver would spread through the sacral canal, the wings of the ilium, and continue to the cranial cavity, by way of the vertebral veins. Thus Batson focused attention on a neglected area of the venous system by recognizing that this venous channel might be a pathway for the near and distant spread of tumor emboli.

After a period of skepticism, many researchers have confirmed the importance of the vertebral, as well as the azygos system of veins in the spread of tumor emboli and infection. It has been conclusively demonstrated in laboratory animals that the vertebral veins are a route of tumor spread following injection of viable tumor cells into the femoral vein. Such spread occurs following ligation of the inferior vena cava and the azygos vein or following abdominal compression alone. Further recent work has been extended to the living adult.

The author reports a series of 40 sequential angiocardiographic injections of the saphenous or femoral vein, all without abdominal pressure, in which opacification of the vertebral and azygos venous systems was demonstrated in about two-thirds of the cases. All cases were between the ages of twenty-six days and four years. Analysis of the entire series gave no reason why vertebral vein opacification was noted in two-thirds of the cases but not in the other one-third.

The normal anatomy of the vertebral and azygos systems is described and illustrated following which the author presents excellent roentgenograms of several of the more common variations in systemic venous return. These include bilateral superior venae cavae, superior vena caval obstruction, persistent left inferior vena cava and absent inferior vena cava. The roentgenograms clearly show the free communication of the systemic veins with the azygos and vertebral veins and thus add support to Batson's concept that the direction of blood flow in these veins may be reversible and may be altered by changes in intrathoracic or intra-abdominal pressure. These variations in venous return which ordinarily receive little clinical emphasis can become extremely important in angiocardiography and certain surgical procedures on the inferior vena cava and portal vein.—*Edward B. Best, M.D.*

General

Birkhäuser, H. Fünf weitere Fälle von Morbus Boeck-artigen Läsionen nach BCG-Impfung. (Five additional cases of Boeck sarcoid-like lesions after BCG-vaccination.) *Schweiz. med. Wchnschr.*, Nov., 1957, *87*, 1434–1439. (From: Tuberkulose-Fürsorgestelle, Basel, Switzerland.)

Chest roentgenography in 5 patients (2 females, ages twenty-five and nineteen, and 3 males, ages eighteen, twenty-one, and twenty-three) revealed transient changes, including hilar enlargement, increased perihilar or interstitial markings, and/or miliary parenchymal elements, which were detected from three months to three years after the performance of percutaneous BCG inoculation. At the time when these roentgenographic changes appeared, 4 of these 5 patients were tuberculin-negative, 3 had modified sedimentation rates, 2 had familial history of tuberculosis, and 1 was in the acute phase of erythema nodosum. In 3 cases, remission occurred spontaneously, while the other 2 received antituberculous chemotherapy, but the disappearance of the roentgenographic findings was allegedly much faster than one would have expected in the presence of acid fast infection. At the time of publication, the chest roentgenograms of all 5 patients were stated as being "unremarkable."

To this abstractor, the causal relationship between the BCG and the reported roentgenographic changes is far from being satisfactorily established. Nevertheless, the author seems to be another step ahead, and declares (cryptically, and without further elaboration) that these occurrences may shed a significant light on the etiology of Boeck's sarcoidosis.—*E. R. N. Grigg, M.D.*

MARGULIS, A. R., FEINBERG, S. B., LESTER, R. G., and GOOD, R. A. Roentgen manifestations of congenital agammaglobulinemia. *Radiology*, Sept., 1957, *69*, 354–359. (Address: R. G. Lester, 412 Union St., Minneapolis 14, Minn.)

Agammaglobulinemia, a relatively new disease, is characterized by four cardinal features: increased susceptibility to infection, absence of gamma globulin from the blood and tissues, absence of circulating antibody from the blood and tissues, and failure of antibody production in response to antigenic stimulation.

Three forms of this disease have been recognized. The first is a congenital disease in which the basic disturbance appears to be an inborn error of metabolism transmitted genetically as a sex-linked recessive trait in males. The second is an acquired form of agammaglobulinemia in which the patients begin to suffer from recurrent episodes of bacterial infection after previous normal health. The third form is a transient agammglobulinemia which appears to be merely an extension of the normal relationships in gamma globulin metabolism. It occurs when the decline in serum gamma globulin concentration which is characteristic of the neonatal period is extended significantly beyond the time at which normal production ordinarily begins and increasing concentrations are observed. An interesting finding in these cases has been the observation that while resistant or recurrent bacterial infections are a fundamental feature of agammaglobulinemia these patients seem to develop an immunity to certain virus infections.

The authors report the roentgen findings in 7 male patients with congenital agammaglobulinemia. All showed extensive pulmonary parenchymal disease with a paradoxical absence of hilar lymph node enlargement. A striking deficiency of nasopharyngeal lymphoid tissue, in spite of recurrent upper respiratory tract infections, was seen. Evidence of sinusitis was frequently found.

The diagnosis can be established only by repeated electrophoretic analyses of the serum, immunochemical determination of serum gamma globulin concentration, and demonstration of the immunologic handicap by studies following extensive attempts at immunization. The roentgen findings, however, can suggest the presence of this disease and it should be thought of when repeated bouts of pulmonary disease, without hilar lymph adenopathy, and the absence of adenoid tissue on roentgenologic examination are found.—*Donald N. Dysart, M.D.*

SCHIMMELPENNING, G. W. Klinischer Beitrag zur Symptomatologie der Phakomatosen. (Clinical contribution to the symptomatology of phakomatosis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Dec., 1957, *87*, 716–720. (Address: Univ.-Nervenklinik, Münster i. W. Germany.)

Phacomatosis is a certain congenital dysontogenetic process primarily of ectodermal layers (skin, brain, eye), but to some extent also of mesodermal and entodermal layers. Included in this syndrome are neurofibromatosis, tuberous sclerosis and von Hippel-Lindau disease, more recently also the Sturge-Weber disease.

The author describes the case of a female, age seventeen, with cerebral spastic convulsions, systemic nevus sebaceus of the left portion of the scalp, a vascular nevus on the left side of the neck together with a tumor and a coloboma of the left upper eyelid. Of special interest from a roentgenologic standpoint is an extensive monostotic fibrous dysplasia of the left hemicranium. This latter finding is fairly characteristic of the Jaffe-Lichtenstein syndrome, the only difference being the unilateral involvement and absence of interspersed cystic areas in this case. The findings also differ somewhat from previously described cases, but are sufficiently characteristic of the syndrome of phacomatosis. Because of certain similarities the following conditions have to be excluded: neurofibromatosis, tuberous sclerosis, neurocutaneous angiomatosis and the Albright syndrome.—*Ernest Kraft, M.D.*

RADIATION THERAPY

PATERSON, RALSTON. The introduction of the rad in radiotherapy practice. A symposium. I. Radiation units in use in Great Britain. *Brit. J. Radiol.*, 1956, *29*, 353–355. (From: Christie Hospital and Holt Radium Institute, Manchester, England.)

Clinicians should ask the physicists: (1) Highest possible accuracy; (2) reproductability of calibrations and (3) units of radiation to be equal throughout the world. In switching to rads for the 4 mev. linear accelerator at Manchester, it was found that the amount of radiation called 1,000 r as determined by a late 1930 calibration was rated as 930 r by a 1954 calibration at National Physics Laboratory. Other centers noted similiar changes. A simple conversion of treatment dosage by letting 93 old r equal 100 new r would suffice except at the time of writing, physicists were not in agreement as to the accurate relationship. An "Exchange Rate" table shows that other centers have found different ratios in calibrations prior to 1952 to the present calibrations. There is an urgent need for the physicists to come to agreement and to establish a definite relationship between the rad and the roentgen.—*Waldo DeVore, M.S.*

GRAY, L. H. The introduction of the rad in radiotherapy practice. A symposium. II. The transition from röntgen to rad. *Brit. J.*

Radiol., 1956, *29*, 355–358. (From: British Empire Cancer Campaign Research Unit in Radiobiology, Mount Vernon Hospital, Northwood, Middlesex, England.)

The rad is not just an improved version of the roentgen. They are 2 complementary units which define different aspects of radiation. The roentgen dose in any volume is defined by the number and energy of photons passing through the element during exposure. The rad dose is the energy imparted by the ionizing particles to the tissue within the volume. The biologic change due to exposure depends on energy absorbed rather than energy passing through. Response is thus related to the rad.

The roentgen is suitable for use in measuring the output from a source but should not be used to represent the unit for the absorbed dose. If dose is given in terms of energy, calculation is simple, as for beta emitting isotopes. If the information is in terms of ionization, a constant is necessary to determine energy expanded by ionizing particles. It is also necessary to know the stopping power ratio between air and tissue. Routine practice of dosimetry using recommended values, the ionizing constant and the stopping power ratio would require the measurement of output in roentgens as at present the determination of the absorbed dose in air and the estimation of absorbed dose in tissue. In cases in which different absorbed dosage is produced at points separated by distance comparable to the range of secondary electrons, the preparation of isodose curves becomes somewhat difficult. This occurs in use of supervoltage therapy and in determining absorbed dosage of bone.

It is concluded that no great problem or added burden would result from the adoption of the rad as the standard unit of recording dosage.—*Waldo DeVore, M.S.*

Day, M. J. The introduction of the rad in radiotherapy practice. A symposium. III. The use of the rad in clinical dosage. *Brit. J. Radiol.*, 1956, *29*, 358–367. (From: Radiotherapy Department, Royal Victoria Infirmary, Newcastle upon Tyne, Great Britain.)

The introduction of the rad presents not a new unit but rather a new concept, the absorbed dose. This new concept is necessary for three reasons: (1) Use of radioisotopes; (2) continuous expansion of high energy roentgen-ray generators; and (3) use of ionizing corpuscles such as electrons, protons and neutrons. Absorbed dose concept existed since the introduction of the rep for isotope use. We have been in effect calculating beta-ray dose in rad and converting to roentgens. Also in supervoltage therapy it has been necessary to use absorbed dose rather than simply a specification of roentgens delivered.

Dosage can be expressed in four ways: (1) Ionization in air (roentgens); (2) ionization in an infinitesimal air cavity in the irradiated medium; (3) energy dissipation in the medium (rad); and (4) ionization in the irradiated material itself. Energy dissipation is the simplest and most general.

If the suggestion that the rad be taken as the basic unit of clinical dose is accepted, several steps will be necessary. In the medium voltage roentgen therapy measurements will continue to be made in roentgens and the physicist will relate tissue absorbed doses in rads to air doses in roentgens. Corrections must be made for the differences in the standardization of dosemeters by the National Physics Laboratory. The Greek letter rho is defined as the ratio of dosemeter correction factor of recent NPL calibration to factor of prior calibration. This is the ratio by which doses at any center should be numerically increased to conform to the new calibrations. In general British radiotherapists should numerically increase their prescribed deep therapy doses by an average of 9 per cent.

In treatment planning, percentage depth dose curve and isodose curves remain unchanged for practical purposes. At present tissue doses in rads are determined assuming that the tissues are water-equivalent, bearing in mind that bone introduces a factor of 1.5 to 2.5. The standard output of each roentgen-ray machine is measured in roentgens and the backscatter factor is replaced by a surface dose factor. A special calculator for exposure has been worked out using the output rate and the surface dose factor.

For high energy radiations the free-air chamber becomes impracticable and absolute dosage is measured by cavity ionization chambers. It is important to consider the relation between the incident radiation flux, the dose and the ionization in the chamber. Chemical methods of dosimetry are also considered. Introduction of the rad to isotopes presents no new problems.—*Waldo DeVore, M.S.*

Ellis, F. The introduction of the rad in radiotherapy practice. A symposium. IV. Conversion of röntgens to rads. *Brit. J. Radiol.*, 1956, *29*, 367–369. (From: Radiotherapy Department, The Churchill Hospital, Oxford, England.)

The rad should influence our thinking and not be merely regarded as a conversion factor. The roentgen exposure is virtually unaffected by the presence of bone but the dose in rads may be much higher numerically because of the presence of calcium. To not make use of the rad is likely to obscure our thinking and to retard the correlation of biologic effects with dose. To change from roentgen to rad for prescription purposes, two things must be remembered: (1) The change in the NPL standard; and (2) the

conversion factor (roentgen/rad), which will vary according to value of W used.

The same prescribed number of rads as have been prescribed in roentgens should give the same clinical results since dosage has been based on practical clinical experience. Errors introduced by so doing will not likely cause much confusion as clinical observations on which prescribing is based are subject to considerable inaccuracies. These are due to physical inaccuracies within one center, between centers and other errors of the treatment itself.

It should be of help to both the radiotherapist and the physicist to have a treatment summary that would allow realization of possible range of dosage absorbed.—*Waldo DeVore, M.S.*

Karnofsky, David A., Golbey, Robert B., and Pool, John L. Preliminary studies on the natural history of lung cancer. *Radiology*, Oct., 1957, *69*, 477–488. (Address: D. A. Karnofsky, 444 E. 68th St., New York 21, N. Y.)

The authors have studied the records of 80 patients with proved pulmonary cancer who were autopsied at Memorial Hospital, New York, in order to try to establish the life history and pattern of growth of cancer of the lung. Certain patterns could be suggested but due to the limited number studied and deficiencies in the records no figures worthy of statistical analysis could be determined.

Three distinctive clinical patterns seem to be present. Type 1.—Clinically localized disease: The cancer tends to grow and extend within the lung. Peripheral metastases may occur but only in the terminal stages. Type 2.—Clinically early generalized dissemination: The cancer produces early evidence of distant metastases to the peripheral lymph nodes, bone, skin, liver, brain and occasionally other tissues. Type 3.—Organ specific distant metastases: This type of cancer seems to grow particularly well in distant specific organs. These secondary lesions occurring either in the brain, bone or liver may appear early and represent the initial complaint.

The authors feel that, if acceptable patterns can be evolved, they may be utilized to demonstrate a patient's clinical status and disease pattern in a simple, graphic manner at the moment that treatment is begun. Several charts are reproduced which show the method of study of these cases.—*Donald N. Dysart, M.D.*

Buschke, Franz. Roentgen therapy of carcinoma of the lung. *Radiology*, Oct., 1957, *69*, 489–493. (Address: University of California Medical Center, San Francisco 22, Calif.)

There are 2 clinical forms of carcinoma of the lung, based on the biologic behavior of the disease, the symptomatology and the therapeutic possibilities.

The first group comprises the undifferentiated carcinomas, which include the oat-cell and the small-cell carcinomas. These tumors behave clinically almost like lymphoblastomas. They are unsuitable for surgery because of their biologic character. They are likewise usually not controllable by radiation, in spite of their radiosensitivity, but some of their symptoms can be palliated by roentgen therapy. A moderate dose—around 3,000 r to 4,000 r in about four weeks—usually produces relief of troublesome symptoms.

The second group of lung cancers consists of the epidermoid carcinomas and the adenocarcinomas originating in the bronchial mucosa. These 2 types are very similar in radioresponse and character of the disease. They progress very slowly and, if they are found in the presymptomatic stage, are surgical problems. When the disease has progressed to the stage of bronchial obstruction, surgical removal is usually impossible. They are also not then amenable to curative roentgen therapy, although a few cases of permanent control by radiation therapy are recorded in the literature. The main indication for palliative roentgen therapy is to reopen the obstructed bronchus and to prevent or postpone the complications secondary to the obstruction. These tumors require a higher dose than those in the first group.

The author bases his report on the observations in 33 patients who received a complete therapeutic course of irradiation delivered by a 2 mev. therapy machine. Six patients were alive six months after treatment and 3 were alive more than twelve months after treatment. He concludes that undifferentiated carcinoma of the lung should be treated if marked symptoms such as mediastinal embarrassment or gross hemorrhage are present. The field should cover the entire mediastinum with a moderate dose—about 3,000 r to 4,000 r—in mid chest. Epidermoid carcinoma and adenocarcinoma should be treated by irradiation only if found to be inoperable, for the effect on specific symptoms which are present or anticipated in the near future. With accurate localization and small fields, tumor doses of 6,000 r can be delivered in forty-five to sixty days, by means of supervoltage therapy. This probably will not result in a significant improvement in results, but better palliation is obtained with less morbidity than by medium voltage therapy.—*Arno W. Sommer, M.D.*

Schulz, Milford D. The results of radiotherapy in cancer of the lung. *Radiology*, Oct., 1957, *69*, 494–498. (Address: Massachusetts General Hospital, Boston 14, Mass.)

This paper is based on a study of 772 patients with cancer of the lung seen during the period 1940 to 1953. Of this group, 148 patients (19 per cent) received no treatment at all. Three hundred and eighty-

five (50 per cent) received only roentgen therapy, while 163 (21 per cent) were treated surgically. Seventy-six, or 10 per cent, were treated by surgery followed by irradiation. It should be recognized that those who were treated only by surgery had localized and resectable lesions. Those who were treated only by irradiation included patients who were deemed unsuitable for surgery or in whom surgery consisted only of biopsy. Those who were treated by surgery followed by irradiation were patients in whom late recurrence in the chest was found after a period of freedom from the disease following surgery. Also in this group were those who had incomplete removal of the tumor because of extension to the chest wall, pericardium, diaphragm, lymph nodes or other mediastinal structures.

Of those treated by roentgen therapy alone, 65 per cent were dead within six months after the beginning of treatment, 85 per cent in one year and all but 5 per cent in two years. Four patients (1 per cent) survived more than five years. The calculated median survival time of this entire group was only four months compared to that of about three months of those who received no treatment at all.

Survival of those who were treated by roentgen therapy following surgery was more favorable. Fifty per cent were alive after one year and 27 per cent survived two years. These figures are similar to the survival rates when surgery alone was the treatment. The median survival time of the 2 groups was calculated to be around twelve months—about three times that in the patients who were treated by roentgen therapy alone or who received no treatment at all.

Analysis of the results indicates that any treatment appears to alter only the early course of the natural history and progression of lung cancer. Excessively large tumor doses did not lead to any significant improvement in the length of survival. One thing stated by the author, however, is the important factor of palliation produced by roentgen therapy. The relief of pain, cough, blood spitting and the prevention or resolution of the distressing complication of superior mediastinal obstruction are very real to the individual patient. The case histories of the 4 patients who survived more than five years are presented.—*Donald N. Dysart, M.D.*

Roswit, Bernard. Present status of chemotherapy of bronchial cancer. *Radiology*, Oct., 1957, *69*, 499–507. (Address: VA Hospital, 130 W. Kingsbridge Road, Bronx 68, N. Y.)

Patients with cancer of the bronchus sooner or later present problems in management of advanced and generalized neoplastic disease. Although radiation therapy will continue to remain the principal palliative method, the polyfunctional alkylating compounds, notable nitrogen mustard, have proved to be valuable aids in relieving distress and disability when the former is no longer effective or feasible.

The author reports his experience with the use of nitrogen mustard in more than 150 patients with widely generalized disease, radiation resistance, exhaustion of radiation tolerance, intractable radiation sickness, and superior vena cava obstruction. Subjective benefits, observed in about two-thirds of the cases, consist in temporary relief of pain, cough, dyspnea, fever and hemoptysis. Objective results, seen in about one-third of the cases, include decrease in size of pulmonary and metastatic lesions, resorption of pleural fluid, re-aeration of atelectatic lung, decrease in neurologic signs, and improvement of the superior vena caval obstructive phenomena. Remissions ranged in duration from one to seventeen weeks after a course of HN_2. Dose levels of 0.4 mg. per kilogram of body weight per course were employed in 1, 2, or 4 fractions, injected directly into the tubing of a running infusion.

Radiation-induced remissions for local metastatic disease are far more lasting than those obtained with HN_2. In the author's series of 38 patients with superior vena caval obstruction, remissions induced by mustard rarely lasted longer than three to eight weeks, while those induced by radiation averaged fourteen weeks. He recommends prompt use of intravenous HN_2, followed in one week by roentgen treatment in daily tumor doses of 150 r until 3,000 r to 4,000 r has been delivered.

Pleural effusion in bronchogenic carcinoma has responded to the intrapleural administration of HN_2 in about 50 per cent of the cases. Results have been similar to those obtained with Au^{198}, P^{32} or Y^{90}. Treatment with nitrogen mustard has the advantage of being less expensive and less hazardous; it is always available, and requires no special facilities. Weisberger *et al.* recommend combined use of radioactive colloids and HN_2. The intra-arterial route for HN_2 therapy has been explored by some workers to deliver principal impact upon the tumor-bearing area while sparing the host. This method has been effective in producing a regression in a variety of human neoplasms. Thiotepa, TEM, myleran, nonane and a number of antimetabolites have been found to possess carcinostatic properties. These preparations, used singly or in combination, as well as multicombination chemotherapy used in conjunction with roentgen therapy are being subjected to extensive investigation. Bronchogenic carcinoma has been selected as one of the principal targets of a cooperative investigation in chemotherapy, employing carefully planned protocols, critical selection of patients and controls, collection of quantitative data and use of entirely objective criteria for evaluating final results.—*Arno W. Sommer, M.D.*

Desaive, Paul, Garsou, Julien, and Ghys, Roger. Principes et modalités d'application d'un appareil de télécuriethérapie rotatoire

au cobalt radio-actif $_{27}Co^{60}$. (Principles and practical application of rotating telecurietherapy with radioactive Co^{60}.) *J. belge de radiol.*, 1957, *40*, 442–543. (From: Centre Anticancéreux près l'Université de Liège et Institute Interuniversitaire des Sciences Nucléaires, Liège, Belgium.)

This comprehensive article begins with an extensive review of the history and of the general physical principles of high energy radiations, with special regard to Co^{60}.

The authors' installation consists of two parallel outer steel rings (diameter 318 cm.) and two parallel inner rings (diameter 126 cm.), held together with spokes and crossbars, so as to form a large wheel, embedded in the floor for 108 cm. The 250 curie (in 1954) source, shielded with lead, in an outer steel casing, is affixed to the inner rings of the wheel. A counterweight permits 360° rotation of the entire device, the moving mass weighing 4,400 kg. A table can be introduced into the space within the inner ring, and positioning of the patient is facilitated by two light sources, one of which is projected in the central beam (from the counterpointer), the other being perpendicular to it, so as to indicate the exact center of rotation at the intersection of the light fascicles. Within its shield, the Co^{60} source (actual size: 60 × 60 mm. external measurements, 20 × 40 mm. internal measurements) is moved with the help of compressed nitrogen. With fixed source, the air dose, at 55 cm. focal distance, was in 1954 19 r/min., while during rotation, the axial delivery increased for a cylinder of 4 cm. diameter to 178 per cent, of 8 cm. to 335 per cent, of 12 cm. to 393 per cent, and of 16 cm. to 412 per cent.

Patients were treated with this device since 1955, and the discussion is based on the results obtained in about 50 cases. The usual absence of general reaction and the paucity of skin reactions are desirable features in the irradiation of deep-seated tumors. In these authors' opinion, the best indications for rotating telecobaltherapy were carcinoma of the larynx, the esophagus, and the urinary bladder and "melanotic epithelioma." From the brief histories included, it appears that they use tumor doses as high as 12,300 r_γ through three fixed fields to the left maxillary sinus, in the hope to control carcinoma of the antrum which had recurred after radium implantation (30 mg. for five days); subsequently, the maxilla had to be resected because of inflammatory changes and the histologic examination failed to demonstrate any residual tumor, but the patient succumbed one year later with cerebral metastases. In another case, a fibrosarcoma of the aponeurosis of the biceps, uncontrolled by about 10,000 r of conventional roentgen therapy, telecobalt was applied through one fixed port until additional 11,500 r_γ were given, leading to "practically complete regression" of the lesion. These authors believe that Co^{60} and the super- and megavoltage sources will replace conventional roentgen therapy in all cases of deep-seated lesions, but they also claim that high energy radiations can control even sarcomata and other tumors which so far have been considered radioresistant.—*E. R. N. Grigg, M.D.*

MELLINK, J. H. Further measurements with the Leiden telecobalt unit. *J. belge de radiol.*, 1957, *40*, 544–563. (From: The Radiological Department, The Academic Hospital, Leiden, Holland.)

In 1955 the Leiden home-made telecobalt unit was reloaded with a 71 curie source having an air dose rate of 17.0 r/min. at a distance of 25 cm. from the source. Dose measurements for 5 applicators were performed, and the dependence of the build-up ratio and the depth of the maximum tissue dose on the nature of the "electron filter" was determined for covering plates of aluminum, brass, tin, and lead, respectively. Brass plates 0.5 mm. thick were selected for the "electron filters" and placed 1.0 cm. from the skin, covering the conical hole of the applicator.

The author's dose measurements agreed closely with those predicted theoretically by Lamerton (*Brit. J. Radiol.*, 1948, *21*, 352). For field sizes up to 140 $cm.^2$, the variation of the percentage depth dose with the source skin distance is given very approximately by the "F" factor of Mayneord and Lamerton for all depths and for all source-skin distances between about 20 cm. and 100 cm. This factor is stated thus:

$$F = \left(\frac{f}{60} \times \frac{60 + d}{f + d}\right)^2$$

where f= source-skin distance, and d=phantom depth in cm. For smaller source-skin distances various published results of different investigators appear to be conflicting.—*Arch H. Hall, M.D.*

TAYLOR, GRANTLEY WALDER. The role of the surgeon in advanced cancer. *Radiology*, Sept., 1957, *69*, 341–343. (Address: 101 River Road, Weston 93, Mass.)

The management of the patient with advanced cancer is handled best as a joint enterprise by the surgeon, the radiologist, and the consultant in internal medicine. Certainly a basic requirement is that one individual supervise the patient's care. The surgeon often is selected not only because of prior patient contact in many instances, but also because he can offer certain procedures which may prolong the patient's life and which can make his days more pleasant. One of the first questions facing the surgeon is—Is the disease really advanced and incurable? If so, even ultra-radical surgery is not feasible and should be reserved for a localized, potentially curable disease with the aim of eradication of the disease to

produce a useful, comfortable and enjoyable existence.

Although the situation of a solitary or localized metastatic process must be assayed on an individual basis, alteration of the hormonal environment has become an established procedure in certain types of cancer. The author feels that adrenalectomy is simply an evolutionary phase in hormonal alteration and eventually will be replaced by hypophysectomy.

Among other methods of palliation, débridement can be useful in removing an odorous, gangrenous extremity or a necrotic, ulcerating mammary cancer. Other procedures which often aid in patient care and remove extreme discomfort are decompressive and by-pass procedures on the respiratory, alimentary, or urinary tract, and direct pain-relieving procedures on peripheral nerves and the sensory tracts of the spinal cord.—*Edward B. Best, M.D.*

Schulz, Milford D. Radiotherapy in advanced and incurable malignant disease. *Radiology*, Sept., 1957, *69*, 321–323. (Address: Massachusetts General Hospital, Boston, 14, Mass.)

Radiotherapy has two functions in the management of malignant disease: (1) To control and cure the disease if possible; and (2) to provide palliation where cure is impossible. The aim must always be complete cure unless it is certain that palliation is all that can be accomplished. Failure to approach a curable situation in a manner likely to produce a cure is even worse than to persist in curative efforts when there is no chance for a cure. The primary purposes of palliative radiotherapy are to provide symptomatic relief and to prolong life. Carefully planned palliative radiotherapy can do much to prolong the cancer patient's useful, comfortable existence. Good judgment, an understanding of disease processes and a knowledge of what can and can not be accomplished by radiotherapy are required. There is a growing tendency for curative radiotherapy to be carried out in larger centers where there are concentrations of highly trained personnel and the expensive equipment and facilities which modern radiotherapy requires. Palliative radiotherapy does not require an elaborate and expensive array of equipment and must remain available to patients close to their homes.—*Arno W. Sommer, M.D.*

Schmitz, Herbert E., and Isaacs, John H. Complications of advanced cervical cancer and their management. *Radiology*, Sept., 1957, *69*, 324–329. (Address: H. E. Schmitz, 55 East Washington St., Chicago 2, Ill.)

Cervical cancer deaths at the Mercy Hospital Institute of Radiation Therapy, from 1933 through 1955, were reviewed by the authors. As a result of a thorough study of these 350 cancer deaths, certain criteria were established for the management of the terminal case.

Pain is a major problem in the management of advanced cervical cancer. The mechanism of pain production by cancer includes compression and destruction of nerves, invasion of contiguous pain-sensitive structures, infiltration and occlusion of arteries, veins and lymphatics, infection and inflammation, and obstruction of hollow viscera. The most effective means of combatting these complications has been palliative irradiation. In the reported series, 187 patients received secondary irradiation with 1,000 kv. roentgen rays or radium or a combination of both. The length of life following irradiation varied from one to forty-eight months. The average survival was 11.6 months. Some patients have lived well beyond the five year interval. Patients not benefited by radiation therapy are sometimes relieved by other means. Extensive palliative operations were carried out in 28 cases. The average survival following surgery was nine and one-half months. In most instances there was symptomatic relief and prolongation of life.

When irradiation is contraindicated or ineffective and radical surgery is unwise, certain procedures may be employed to interrupt pain pathways. This may be accomplished by neurolytic agents, chordotomy or frontal lobotomy. Besides these specific measures, maintenance of adequate nutrition, correction of anemia and avitaminosis and attention to the morale of patients are of prime importance.—*Arno W. Sommer, M.D.*

Silverstone, Sidney M., and Melamed, Jack L. Effective irradiation with radium for cancer of the cervix. *Radiology*, Sept., 1957, *69*, 360–371. (Address: S. M. Silverstone, 945 Fifth Ave., New York 21, N. Y.)

The authors present information in the form of isodose contours of the radium system employed in the Mount Sinai Hospital. The basic plan of radium therapy of cancer of the cervix uteri consists of the application of a tandem of 3 tubes in the uterine cavity and a colpostat of 2 ovoids in the vaginal vault. The tandem contains 40 mg. of radium filtered by 1 mm. of platinum and arranged in the 3 tubes in strengths of 15—15—10 mg. from fundus to external os. The colpostat contains 50 mg. of radium filtered by 1 mm. of platinum, 25 mg. in each ovoid. Ovoids are placed in the lateral fornices, with the maximum separation consistent with safe distensibility of the vaginal vault. The representative point system is employed for the calculations of dosage in the pelvis.

The photographic method described by Loevinger and Spira was employed by the authors to construct the isodose contours. In this method, slow-acting (Adlux) film is exposed to the radium in the tandem and colpostat placed in a pressdwood phantom, so

as to reproduce the actual conditions in the female pelvis. Within a certain range, the resultant photographic densities bear a linear relationship to the radium exposure. By means of a densitometer, the photographic densities at Points A and B may be traced on the exposed film, and isodose contours thus constructed. The isodose contours through Point A, Point B, and a point in the parametrium midway between these in respect to dosage represent volumes of tissue corresponding approximately to cervical cancer Stages I, III and II respectively.

A set of diagrams, indicating the isodose contours for various arrangements of radium, is presented.—*Arno W. Sommer, M.D.*

Testa, Elsie R. Method of dosage calculation for linear radium sources. *Radiology*, Oct., 1957, *69*, 558–563. (Address: Bellevue Hospital Center, First Ave., and 27th St., New York 16, N. Y.)

A simplified, rapid method for calculating intracavitary radium dosage is presented, particularly as it applies to the cervix and uterine cavity. With a radium source in place, an anteroposterior roentgenogram is taken centered directly over the radium. The tube is then shifted a known distance (4 in.) and a second exposure is made on the same film. A constant target-film distance of 40 inches is maintained. Knowing the target-film distance, the tube shift, the image shift on the film, and certain anatomic relationships of the point of interest, as for example Point A, the geometric relationship between the two ends of the radium source and the point of interest can be established. A triangle is then constructed to show this relationship. The distances thus obtained are used in determining dosage, according to the Quimby linear source tables.

The method was tested by constructing models of radium implants and the calculated distances checked against actually measured distances. The error was in the neighborhood of 5 per cent.—*Z. Petrany, M.D.*

Barnes, D. W. H., Corp, M. J., Loutit, J. F., and Neal, F. E. Treatment of murine leukaemia with x-rays and homologous bone marrow; preliminary communication. *Brit. M. J.*, 1956, *2*, 626–627. (From: M. R. C. Radiobiological Research Unit, A.E.R.E., Harwell, Berks, Great Britain.)

Normal mice will survive lethal doses of roentgen rays if subsequently injected with normal bone marrow from either their own or an homologous strain.

Lymphoid leukemia was transmitted to selected groups by injection of cells from affected animals. Death of all "takes" is certain within two months and previous experiments have shown transmission to be nearly 100 per cent effective.

Following irradiation (half-value layer 1.2 mm. Cu) with 1,500 rads in twenty-five hours, and subsequent injection of isologous marrow, 25 or 35 subjects have survived three months. Lesser amounts of radiation in shorter times produced no cures.

Total destruction of 100 per cent of the leukemic cells is necessary for cure if the hematopoietic tissues are repopulated by bone marrow from the same strain. Whether homologous myeloid tissue will retain the capacity to destroy a certain number of surviving malignant cells without harm to the host must be determined.

Experiments showing promise of the successful treatment of leukemia in any animal deserve the fullest attention and encouragement.—*Dale Undem, M.D.*

RADIOISOTOPES

Bauer, Göran C. H., and Ray, Robert D. Kinetics of strontium metabolism in man. *J. Bone & Joint Surg.*, Jan., 1958, *40-A*, 171–186. (From: The University of Lund, Department of Orthopaedics, Malmö, and the Division of Orthopaedic Surgery, University of Washington Medical School, Seattle, Wash.)

The metabolism of strontium in man has been investigated using strontium 85, a gamma emitter with 65 days half-life, in view of the similarity between the skeletal metabolism of calcium and strontium and the potential health hazard of strontium 90.

Five normal orthopedic patients each received 30 μc of Sr^{85} intravenously. Serum activity was measured immediately following injection and at intervals over a period of seven to fourteen days; urine and fecal activities were also determined. In addition, the radiation was counted externally at intervals over patella, femur and tibia as well as at eight positions around the trunk at each of six levels between suprasternal notch and pubis. The variation of serum activity with time is expressed as the sum of four descending exponentials for which the constants are derived. The renal clearance-rate showed little variation over the period of the experiment, while intestinal excretion accounted for about 20 per cent of the total and at the end of five days the total retention was about 45 per cent of the injected dose.

External counting over the thigh (muscle+bone) showed a very rapid rise within minutes following injection in contrast to the knee count (bone) where the maximum is reached after some twenty-four hours; in both cases the subsequent decrease in activity is considerably slower than for serum. The torso survey shows a progressive change from an initial isotropic activity to a marked concentration over bone after thirteen days.

The results of this and previous studies suggest a four compartment model for the strontium space

with two outlets, representing removal of strontium by accretion to new bone formation and excretion. With an electrical analogue of this model the rate constants can be determined and the effect of variations in the parameters studied. Strontium appears to divide into a small exchangeable fraction and the larger fixed skeletal component; a possible interpretation of the kinetic model would identify the compartments with serum, extravascular-extracellular fluid, intracellular fluid and bone strontium. In general, agreement with the experimental values is satisfactory but the limitations of the analogue are outlined and it is emphasized that many other models consistent with the experimental data may be devised of which alternatives are being studied by the authors.—*Raymond S. Quick, B.S.*

Matuska, R. A., Hahn, P. F., Carlson, R. I., Auerbach, S. H., and Meneely, G. R. Further observations on the lymphatic pick-up of radioactive silver-coated gold colloid administered intrathoracically to dogs. *J. Thoracic Surg.*, Jan., 1958, *35*, 135–138. (Address: R. A. Matuska, 4247 Hamilton St., Cincinnati, Ohio.)

The authors report on the result of intrapleural and intramediastinal injection of radioactive silver-coated gold colloid in dogs. Ten dogs received at least 50 mc of silver-coated radioactive gold intrapleurally after pneumonectomy. In a second experiment, 12 dogs received injection of 45–51 mc of the isotope into the mediastinum under direct vision. In the third experiment, 10 dogs were subjected to lobectomy and each received 35–50 mc of the isotope in the pleural space. Study of the mediastinal lymph nodes by histologic means and a Texas well counter revealed a consistent radioactive effect on the mediastinal lymph nodes. No harmful effects were noted following intrapleural administration of the isotope. The incidence of bronchial stump dehiscence following intramediastinal injection of the colloid in pneumonectomized dogs was 25 per cent.

Six patients have been treated with radioactive silver-coated gold following pneumonectomy for bronchiogenic carcinoma at the Thayer Veterans Administration Hospital. It is not thought likely that lymph nodes replaced by tumor would pick up sufficient radioactive material to destroy them, but it seems possible that small tumor emboli in these lymph nodes could be destroyed by this method. No ill effects in the cancer patients have been noted from the therapy. Dosage has ranged from 75 to 150 mc. Three of these 6 patients are alive, eight, twenty, and twenty-seven months following therapy without evidence of recurrent tumor.—*Paul C. Ofelt, M.D.*

Rubin, Philip, Brace, Kirkland C., Gump, Hazel, Swarm, Richard, and Andrews, J. Robert. The radiotoxic effects of S^{35} in growing cartilage; consideration of radioactive sulfur (S^{35}) as a possible radiotherapeutic agent in chondrosarcomas. *Radiology*, Nov., 1957, *69*, 711–719. (Address: P. Rubin, Strong Memorial Hospital, 260 Crittenden Blvd., Rochester 20, N. Y.)

It has been shown in rats that the radioactive sulfur of labeled sulfate is incorporated into chondroitin sulfate and fixed in cartilage. This has suggested the possibility of utilizing this isotope in the treatment of humans with chondrosarcoma. In order to obtain evidence that a selective radiotoxic effect in cartilage was present, experiments were conducted on 11 litters of suckling rats.

The results of these experiments showed that the minimal dose of S^{35} required to arrest cartilage growth was 0.1 mc/gm. of body weight. The changes were severe, resulting in an irreversible alteration and destruction of the cartilage in the metaphyses and epiphyses. Doses of 0.1 mc/gm. given three times at biweekly intervals to a total amount of 0.3 mc/gm. resulted in more profound changes in cartilage growth when 0.3 mc/gm. was given in a single injection. The maximum tolerated levels compatible with survival were in the neighborhood of 0.3 mc/mg. as a single injection, but, when this same dose was given by a fractionated schedule, it was better tolerated and no deaths occurred. This fractionated dose of 0.3 mc/gm. was also more effective in arresting and altering cartilage growth and less toxic systemically than a single injection of 0.3 mc/gm.

Now that these facts have been established in animals, it must be determined if they will apply also to patients with chondrosarcoma. Elaborate tracer studies with multiple serial biopsies are being done. If the tracer tissue assays show a favorable differential ratio of S^{35} between the neoplastic and other normal tissues, it may be possible to find a method of treating patients with radiosulfur in such a way that the neoplastic cartilage will be destroyed without causing undue toxicity in the patient.—*Donald N. Dysart, M.D.*

Gross, W., Wingate, C., and Failla, G. Determination of disintegration rate for gamma-emitting isotopes. *Radiology*, Nov., 1957, *69*, 699–703. (Address: W. Gross, Radiological Research Laboratory, 630 W. 168th St., New York 32, N. Y.)

A method is described for obtaining disintegration rates of gamma-emitting isotopes from the rate of energy transfer to a surrounding medium as indicated by ionization measurements in a series of concentric spherical shell ionization chambers of varying gap widths. The method offers a more convenient means of determining the disintegration rate of gamma-emitting, K-capture isotopes than the more conventional methods. It is accurate, involves small

corrections, and does not require a strong source. A special type of chamber, developed by White, Marinelli, and Failla was used in this study.

This report deals with the preliminary tests of accuracy of the new method performed with a gold-198 source, the disintegration rate of which is known by other methods.—*Arno W. Sommer, M.D.*

MISCELLANEOUS

Tievsky, George. Ionizing radiation and a sense of proportion. *J.A.M.A.*, April 5, 1958, *166*, 1667–1672. (Address: 1739 Eye St., Washington, D.C.)

Considerable alarm has been produced by the lay and scientific press regarding the harmful effects of ionizing radiation. All types of radiation exposure including medical, industrial, background and fallout from nuclear bombs are under scrutiny as a result.

The genetic effects as well as injury to the individual have been emphasized. Numerous figures have been given as to the amount of radiation an individual can be "safely" exposed to and the amount that would cause injury to the genes. The National Research Council estimated that a gonadal dose of 10 r (which covers a wide margin of safety) for large populations should not be exceeded in the reproductive age group. It should be emphasized that this does not pertain to an isolated individual. It has been estimated that 4.3 r comes from natural radioactive background sources, 0.1 r from fallout from nuclear bomb explosions and 4.6 r from medical and dental sources.

The maximum permissible dose for radiation workers was set at 0.3 r per week. Film badge service was recommended for those using ionizing radiation. It was emphasized that all physicians using radiation equipment should conform to the recommendations in the NCRP Handbook 60.

It has been recommended that radiation should be confined to the smallest possible area and that there should be a reappraisal of all routine examinations, the use of radiation therapy in benign conditions and the use of radioisotopes. Radiation procedures in children and pregnant women should be done with special care. A personal record of radiation received is not practical as yet.

Physicians must regard the hazards of radiation with the same rational thinking as other hazardous procedures in medicine are regarded. The degree of hazard must be weighed against the benefit for the individual. Laymen and nonmedical scientists cannot make this judgment. This must be recognized by the scientists outside the medical field and made clear to the lay public.

There is no reason for the practitioner to feel uneasy about radiation when it is being used with conservative judgment and skill.—*Charles W. Cooley, M.D.*

Richards, Albert G. Roentgen-ray doses in dental roentgenography. *J. Am. Dent. A.*, March, 1958, *56*, 351–368. (From: School of Dentistry, University of Michigan, Ann Arbor, Mich.)

A study was made to determine the effect of various technical factors upon the dose of radiation that dental patients receive. To compare exposures of one technique to another it was necessary to standardize a method of comparison. The experimental procedure is described.

It was found that exposures at 90 kv. required only 36 per cent as much time as at 65 kv. The use of filters increases the exposure time but reduces the skin dose received by the patient. Scattered radiation increases with higher voltage and decreases with filtration and with use of a lead diaphragm. Backscatter is found to increase with increasing filtration. Very little increase in scattered radiation is obtained by using large exposure areas, thus the function of the diaphragm is to limit the area of skin exposed. Target skin distance has no effect on the amount of backscatter for constant air dose. Both skin dose and depth dose are increased for added filtration and higher voltage.

Studies of radiation dose to the gonads revealed that no radiation is scattered through the body to the gonads. Using higher voltage and filtration reduced the gonadal dose in adults by 12 per cent and the use of diaphragms, high energy developer and ultra-speed film reduced the gonadal dose in the same proportion as the reductions in exposure time. Longer target to skin distances offered no variation in gonadal dose.

It is concluded that under the proper conditions, a dose of as high as 23.8 r is delivered to the patients' jaws with only 2.1 mr reaching the gonads. It is recommended that changes be made to further decrease the radiation to the patient by using in the following order: (1) Increased filtration; (2) smaller diameter beam; (3) ultra-speed film; (4) high energy developer; and (5) higher kilovoltage.—*Waldo DeVore, M.S.*

Young, Barton R., Funch, Robert B., and MacMoran, Jay W. Ultrashort (millisecond) timing and a rapid film changer in pediatric radiography. *A.M.A. J. Dis. Child.*, March, 1958, *95*, 300–304. (From: Department of Radiology, Germantown Hospital, Germantown, Pa.)

Ultrashort exposures have been studied to determine if they are better than conventional exposures. The Dynapulse timer described controls the high voltage circuit rather than the primary. Comparison of timing methods by rotating a wire mesh with synchronous motor at known speeds was made showing that motions of 80 cm./sec. are stopped by a one

millisecond exposure. No differences are noted by timing methods on chest and extremity exposures. It is believed that fewer retakes will be necessary by the millisecond exposures. Comparison of angiocardiograms of dogs did not show the 1/1000 second exposure to be any superior to the 1/120 second exposure. In clinical practice Dynapulse timers have been used principally for cardiac and cerebral angiography and aortography. Dynapulse timing does not offer diagnostic superiority over rapid impulse timing except where rapid motion is a problem.

A rapid film changer is described that features electronic programming that offers reduction of patient exposure.—*Waldo DeVore, M.S.*

ADAMS, GAIL D. An experimental determination of energy absorbed in water from an x-ray beam. *Radiology*, Dec., 1957, *69*, 867–880. (Address: Radiological Laboratory, University of California Medical Center, San Francisco 22, Calif.)

This is a highly technical article of primary value to physicists and radiologists interested in the problem of the measurement of the absorbed dose (rad).

An experimental method is presented for determining absorbed dose in water without formal use of the Bragg-Gray relation. The method employs a calorimeter to measure the energy content of a roentgen-ray beam and requires detailed knowledge of the absorption of most of this energy in a water phantom, considered tissue-equivalent for these purposes. The method is applied to 2 narrow beams of roentgen-rays from a 70 mev. synchrotron. The beam energy requirement to produce ionization is found to be close to expectation. The calibration made for the narrow beams was extended to large fields, for the largest of which the result of importance is 0.86 rad per esu./cc.

The original article must be studied for a detailed description of the experimental method, and an analysis of the theoretical application and experimental results.—*Walter H. Jarvis, Jr., M.D.*

LAW, L. W., and POTTER, MICHAEL. Further evidence of indirect induction by x-radiation of lymphocytic neoplasms in mice. *J. Nat. Cancer Inst.*, March, 1958, *20*, 489–493. (From: Leukemia Studies Section, National Cancer Institute, Bethesda, Md.)

Lymphocytic neoplasms arise spontaneously in the thymus of certain strains of mice and also following treatment with roentgen radiation, carcinogenic hydrocarbons, or estrogens. Total thymectomy greatly reduces the incidence and increases the mean age at death in the spontaneous disease in certain strains of mice. In certain other strains, such as the high leukemic strain, lymphocytic neoplasms appear to arise at extrathymic sites and thymectomy does not influence the incidence of this neoplasm.

In some strains, thymectomy followed by a course of roentgen irradiation and the implantation of a nonirradiated thymus from the susceptible parental strain restored this potentiality. Lymphocytic neoplasms developed in the transplanted thymic tissue as late as twenty-eight days following irradiation. The neoplastic cells arising at the site of the thymic grafts have been shown to be derived from either the nonirradiated cells of the graft or from cells of the individual host which repopulate the grafts. Thus the cells of the lymphoid series that are eventually transformed to the neoplastic state need not receive direct radiation to become neoplastic.—*Charles W. Cooley, M.D.*

ANGER, HAL O. Scintillation camera. *Rev. Scient. Instruments*, Jan., 1958, *29*, 27–33. (From: Donner Laboratory of Biophysics and Medical Physics and Radiation Laboratory, University of California, Berkeley, Calif.)

The spatial distribution of a gamma ray emitting isotope is usually studied by a directional counter coupled to a mechanical scanning system and recording device. This paper describes an alternative method using a gamma ray "camera" in which each pulse initiated by the radiation in a large scintillation crystal triggers a light spot at the correct relative position on the screen of a cathode-ray oscilloscope. The pattern of spots on this screen is photographed for a suitable period of time and generates, by random scanning, a progressively more detailed picture of the radioactive source.

A small aperture in the lead housing of the camera forms a pin-hole through which radiation reaches a 4 inch diameter by $\frac{1}{4}$ inch thick sodium iodide crystal backed by seven 1.5 inch diameter photomultiplier tubes. The relative intensities of the scintillations at the different tubes provide X and Y positioning signals to locate the oscilloscope beam which is triggered when the combined pulses pass a pulse-height selector, so producing a spot on the screen at a point corresponding to the origin of the radiation.

The effect of aperture size and duration of photographic exposure is shown by scintillation pictures of test patterns and a number of typical thyroid distributions are illustrated; these are obtained in twelve to fifteen minutes with doses of 7.5–12.5 μc I^{131}. The factors affecting resolution, sensitivity and distortion are discussed and future possibilities outlined. A block schematic diagram is included but the detailed electronic circuit is not given.—*Raymond S. Quick, B.S.*